The New England
Journal of Medicine

VOLUME 212 MAY 16, 1935 NUMBER 20

ELECTROCARDIOGRAPHIC STUDY OF CASES OF CORONARY OCCLUSION PROVED AT AUTOPSY AT THE MASSACHUSETTS GENERAL HOSPITAL, 1914-1934*

BY HOWARD B. SPRAGUE, M.D.† AND EDWARD S. ORGAIN, M.D.†

RECENT studies of the localization of cardiac infarcts by electrocardiographic control have led us to report the experience of the Massachusetts General Hospital in the cases of coronary sclerosis and occlusion that have come to necropsy in relation to their electrocardiographic findings as recorded by standard limb leads.

It has been fairly well agreed, especially through the work of Barnes and Whitten, Parkinson and Bedford, and Wilson and his associates, that an infarct of the anterior and apical portions of the heart produces electrocardiographic changes of the so-called T_1 type, in which a high origin and later an inversion of the T wave in lead one is associated with a low origin but positive deflection of the T wave in lead three. Infarction of the posterior or diaphragmatic region of the left ventricle results in the T_3 type of curve in which the high origin and late inversion are found in the T wave in lead three. The S-T interval and T wave of lead two tend to be influenced in a similar way as the T wave of the lead showing the inversion. Changes in the Q-R-S complexes, characterized by the development of an initial downward deflection (Q wave) also occur in the lead in which T becomes inverted.

Infarction of the anterior wall of the left ventricle is usually produced by occlusion of the anterior descending branch of the left coronary artery, while that in the posterior wall is commonly associated with involvement of the posterior descending branches of the right coronary. In certain instances the posterior wall of the left ventricle is supplied by branches of the left circumflex artery instead of by the right. The apex of the left ventricle is usually supplied by the anterior descending branch of the left but may receive its blood supply from the posterior descending branches of the right coronary or even from the posterior descending branches of the left circumflex.

Barnes and Whitten summarize their observations of cardiac blood supply as follows: "In the average normal heart the left coronary artery supplies the entire anterior surface of the left ventricle, the adjacent third of the anterior surface of the right ventricle, the apex of both ventricles, all of the interventricular septum at the apex, the anterior two-thirds of the septum above that point and the left half of the posterior surface of the left ventricle. The right coronary artery usually supplies two-thirds of the anterior surface and all of the posterior surface of the right ventricle with the exception of the apex. It also generally supplies the posterior third of the interventricular septum (except at the apex) and the adjacent half of the basal three-fifths of the posterior surface of the left ventricle."

The division of cases of coronary occlusion into the two types is a useful advance in our knowledge of localization of cardiac infarct, and the ability to recognize the region of muscle necrosis is helpful in the diagnosis and perhaps the prognosis of coronary thrombosis. In many instances, however, the application of this classification to clinical cases is difficult and implies a simplification of types which does not exist. We would be inclined to believe that a study of the electrocardiographic variants seen in coronary occlusion is also needed to prevent too much insistence upon the typical tracings of acute coronary occlusion for diagnosis. Work being done by various investigators upon the records taken with precordial leads may give much needed information.

MATERIAL STUDIED

From October 14, 1914 to April 27, 1934 there have been 3889 cases autopsied at the Massachusetts General Hospital. Of these, 131 cases have had a pathological diagnosis of occlusion of some part of the coronary system or some degree of myocardial infarction. Sixty-one of these have had reasonably adequate electrocardiographic study and it is this group which we are reporting*. In spite of some overlapping it seemed most useful to divide them into three main classes—(1) Acute coronary thrombosis, (2) Old coronary sclerosis or thrombosis with additional recent infarct, (3) Old coronary sclerosis with or without evidence of healed infarcts. In surveying material such as this it

*From the Cardiac Laboratory and Cardiac Clinic of the Massachusetts General Hospital.

Read at the meeting of the American Heart Association, Cleveland, Ohio, June 12, 1934.

†Sprague, Howard B.—Assistant Physician, Massachusetts General Hospital. Orgain, Edward S.—Instructor in Medicine, Duke University, 1935. For records and addresses of authors see "This Week's Issue," page 945.

*The series also includes one case whose autopsy material was supplied us by Dr. J. C. Gant.

becomes at once obvious that coronary disease as it is seen in a general hospital is usually a diffuse process whose effects upon various parts of the cardiac wall lead to complicated electrical variations and that even in acute coronary thrombosis we are not dealing with an isolated picture of heart muscle damage such as sometimes occurs in younger cases coming under the eye of the coroner or medical examiner. But since it is a group such as ours that lives long enough to be studied by the practising physician it becomes the more important that we discover what such cases show clinically, by electrocardiogram, and at the autopsy table.

TABLE 1

ACUTE CORONARY OCCLUSION

Location of Infarct	No.		Electrocardiogram Consistent		Inconsistent	Negative	Questionable
Apical or anterior left vent.	9	10	6	7	1	1	1
Apical and lateral left vent.	1		1				
Apical, lateral and posterior left ventricle	1	7		2			1
Posterior (diaphragmatic) left ventricle	4		1			2	1
Posterior and lateral left ventricle	1						1
Right ventricle	1		1				
No. of Cases	17		9		1	3	4

TABLE 2

OTHER ELECTROCARDIOGRAPHIC CHANGES OCCURRING IN ACUTE CORONARY OCCLUSION

Location of Infarct	Low Voltage	Marked left axis deviation	Ventricular Paroxysmal tachycardia	Partial A-V Block	Changing Auricular Pacemaker	Intraventricular Block	Left Bundle Branch Block*	Auricular Premature Beats	Ventricular Premature Beats	Auricular Fibrillation
Apical left ventricle	3	2				1			1	1
Apical, lateral and posterior (left)			1							
Apical and lateral (left)								1		
Diaphragmatic (posterior) (left)				1	1		1			
Right ventricle									1	
No. of cases	3	2	1	1	1	1	1	1	2	1

*The new terminology of Wilson is used throughout this report.

ACUTE CORONARY OCCLUSION

Seventeen cases of fresh coronary thrombosis were selected for study. All had had electrocardiograms within one month of death and all but two within two weeks. In all cases a typical area of cardiac infarction was demonstrated at autopsy. This was in the apex of the left ventricle alone in eight cases; in the apex and lateral wall in one; in the apex and anterior wall in one; in the apex, lateral and posterior walls in one; in the posterior wall in four; in the lateral and posterior wall in one; and in the right ventricle in one. Table 1 shows the correlation between the area of infarct and the electrocardiogram in which the electrocardiogram is called "consistent" when an anterior or apical infarct is associated with the T_1 type of change and a posterior infarct with the T_3 type.

From table 1 it appears that nine of the seventeen cases showed T wave changes typical enough to have permitted localization of the infarct before death, the greatest consistency being in the six cases with T_1 change in nine instances of infarct of the anterior and apical walls of the left ventricle. Of the total number with infarct, ten were essentially anterior, five were essentially diaphragmatic or posterior, one included almost the entire left ventricle, and one all of the right ventricle except the septal wall.

Eight cases showed electrocardiograms which were not satisfactory for localizing the infarct. One was inconsistent with the demonstrated lesion, three were non-diagnostic and four were questionable. Table 2 shows the other electrocardiographic changes in the total group of seventeen cases.

Inconsistent Electrocardiogram. Case A. A., male, aged fifteen. The history of substernal distress in this case was of only one month's duration associated with attacks of nocturnal dyspnea. Autopsy showed numerous old scars of the left ventricle with a fresh infarct of the apex and lower part of the septum. The anterior descending branch of the left coronary was occluded. The right coronary showed moderate sclerosis. Electrocardiogram twelve days before death showed inversion of the T waves in leads two and three of the characteristic shape found in posterior infarction.

Questionable Electrocardiograms (4 cases). W. S., male, aged fifty-eight. For two years the patient had had dyspnea on climbing stairs. Sudden severe substernal pain awoke him from sleep two weeks before death. The course was typical of coronary thrombosis ending with rupture of the heart and cerebral embolus. Autopsy showed the pericardial cavity full of clotted blood. There was a softened area of reddish black discoloration extending from the left border around the left and posterior surfaces of the left ventricle to the septum and involving over half the left ventricular wall. This area bulged slightly and on the upper margin near the left border there was a perforation admitting the tip of a pair of forceps. Both coronaries showed numerous, almost contiguous, atheromatous plaques. The posterior descending branch of the left coronary was partially calcified and narrowed to one half its diameter and completely occluded in one spot by a fresh thrombus 0.5 cm. in length. Electrocardiogram was taken thirteen days before death and showed late deep inversion of T_1. T_2 was flat and T_3 upright. This case has been called questionable in this series because although apparently inconsistent with the location of the infarct there was a possibility of incorrect labeling of the electrocardiographic leads since the summation of the Q-R-S and T complexes was such that lead three may have been incorrectly numbered as lead one.

O. L., male, aged fifty-six. For seven or eight years this man had had dyspnea and substernal tightness on exertion. Four years ago edema appeared. Two weeks before he came under observation the symptoms increased markedly with orthopnea and pain in the right upper quadrant of the abdomen. The course was that of rapidly progressive congestive failure. At necropsy the heart showed probable complete occlusion of the anterior descending branch of the left coronary artery with moderate atheroma of the other branches and fresh infarction of the apex and lower part of the septum. Electrocardiogram taken four days before death showed low voltage and a very slight elevation of the S-T segment in lead one but no characteristic upward convexity or inversion of T_1. There were multiple lung infarcts.

The two other questionable or non-diagnostic cases were complicated by intraventricular conduction defects. One had infarction of almost the entire left ventricle chiefly by involvement of the left circumflex artery and showed ventricular paroxysmal tachycardia. The other had left bundle branch block associated with infarction of the posterior wall of the left ventricle including the apex, with rupture of the heart.

Negative Electrocardiograms (3 cases). (1) H. T., male, aged forty-eight. The patient was troubled for two months by "gasping spells" and for six weeks by substernal pain. Two days after surgical removal of a thyroid adenoma which was thought to be a factor in his symptomatology, the patient died suddenly. At autopsy a softened depressed area was found in the posterior wall of the left ventricle involving almost the entire upper third. On section it was found to be hemorrhagic and necrotic. About 1 cm. from its origin the left circumflex artery gave off a moderate-sized descending branch leading to the infarcted area which was completely occluded by a firm partially calcified thrombus. The heart weighed only 300 grams. An electrocardiogram taken thirty-six days before death showed sino-auricular tachycardia with a diphasic T wave in lead one. Another one recorded one week before death showed only slight left axis deviation.

(2) F. S., male, aged fifteen. This patient complained of attacks of dyspnea and substernal choking on exertion for three years. Two months before he was seen he had had a severe attack lasting one hour. This was repeated on the day before admission to the hospital. The heart at necropsy showed a softened area of infarction 2.5 cm. in diameter on the posterior wall of the left ventricle near the auriculo-ventricular junction. At the periphery there were numerous irregular, purplish hemorrhagic and whitish areas, some extending to the papillary muscles. The outer three-quarters of the wall was most affected. The coronaries showed moderate atheromatous degeneration with calcification. The left circumflex artery was completely occluded 1 cm. from its origin by a pinkish gray thrombus, and the terminal portion of the right coronary 4-5 cm. from the infarcted area was similarly completely occluded. Eight days before death the electrocardiogram showed normal complexes. The T waves in leads one and two were upright and T_3 was slightly inverted. The electrical axis was normal.

(3) B. B., male, aged fifty-three. The patient had typical coronary occlusion three weeks before the electrocardiogram was taken. He died three days later. The heart at autopsy showed a mottled brownish-grey appearance over the entire endocardial surface of the left ventricle with fibrous streaks through the body of the muscle. The anterior descending branch of the left coronary showed an organizing thrombus completely occluding it 2 cm. from its origin. Moderate atheromatous changes were seen in the other coronaries. Electrocardiogram showed only left axis deviation with slightly low origin of T_1.

Comment. This group of seventeen cases of acute coronary closure should more nearly approximate experimental occlusion in animals, and the findings by electrocardiogram should be more readily correlated with the pathological conditions demonstrated at autopsy, than those complicated by multiple infarcts. Even in this group, however, it is difficult to find completely satisfactory material. Accurate localization of the infarct by the electrocardiogram was possible in 53 per cent of the entire group, and in 70 per cent of those in which the infarct was essentially anterior. In the seven which were essentially in the posterior region of the left ventricle or in the right ventricle the electrocardiograms available were diagnostic in thirty per cent. This may be due to the fact that patients with posterior infarcts have a better prognosis and do not come so often to autopsy during the stage of fresh or solitary infarction in this area but in our small series of seven cases it was greatly influenced by the fact that one of the seven had ventricular paroxysmal tachycardia and the other left bundle branch block which made the electrocardiograms non-diagnostic.

Failure to correlate electrocardiographic and autopsy data accurately in these cases is due to many factors immediately obvious to anyone attempting to collect information from past records particularly in a field which is just beginning to be understood with accuracy. This failure should make us more eager in the future to explain the discrepancies. Of course great care must be taken to see that the electrodes are attached to the correct leads and that the tracings are properly labeled, but it is impossible to verify these points in electrocardiograms taken several years ago, although by inspection of the tracing alone it is usually clear what mistake may have been made.

The factors more frequently causing confusion are concerned with other influences in the heart altering the character of the record in coronary occlusion, and the limitations dependent upon routine pathological study of the heart which may not include sufficient care in the observation of details. One of the chief mechanisms in the heart which modify the electrocardiogram is intraventricular block which usually obscures the characteristic Q-R-S and T changes. In some instances, however, fresh occlusion in the presence of intraventricular block will cause marked changes in the electrical axis and the T wave. Ventricular paroxysmal tachycardia also prevents accurate localization of infarct. The effects of hypertension, auricular fibrillation and digitalis at times cause confusion. Other types of myocardial injury not dependent on coronary disease may produce profound changes in the T waves, if not the more sensitive alterations in Q waves. Pericarditis, uremia, rheumatic carditis, diphtheria, rare types of cardiac failure of unknown etiology, and possibly coronary spasm from the use of tobacco should be mentioned in this connection. It also appears that marked involvement of the major part of one ventricle or the other, or of both, may produce uncertain electrocardiographic records. Again, as Wilson has shown in dogs, the effect of the electrical potential changes at the margins of the infarct the extent of which, on examination of the heart may have escaped our accurate delimitation, are more important than those at the center of the necrotic area. One other very important factor in the apparent discrepancy between electrocardiographic and pathologic findings is the failure to record electrocardiograms at the optimum time in the course of the occlusion or to neglect to take serial tracings on successive days during the early stages of infarction.

In the examination of the heart itself are to be found other sources of error. The routine method of opening the chambers of the heart leads to a distortion of the relations, and the extent of infarcts at the apex may not be accurately determined, especially as regards the involvement of the diaphragmatic or posterior surface. In some cases, also, the fresh infarct may be obvious but others deep in the muscle, not involving the endocardial or epicardial surfaces, may be missed. In other cases the coronary vessels may be found patent to a certain degree when the patency is due to recanalization of the vessel leading to a hidden area of infarction. It has been suggested by Wilson that gross transverse sections should be made without opening the heart in the customary manner, perhaps after injection of the coronaries to test their patency, as the best method of finding all the myocardial changes. Such study will add much to our knowledge as further investigation with precordial leads is carried on.

OLD CORONARY SCLEROSIS OR OCCLUSION WITH ADDITIONAL RECENT INFARCT

A series of sixteen cases was studied in which, in addition to an old or slowly developing coronary closure, there was found a fresh throm-

TABLE

OLD CORONARY SCLEROSIS OR OCCLUSION W

No. of Case		Age and Sex	Coronaries Involved	Location of Old Infarct
1	J. B.	M 56	Much sclerosis of right coronary. Fresh occlusion anterior descending branch of left.	Upper part posterior wall left ventricle.
2	N. O.	M 62	Left circumflex almost occluded and anterior descending entirely occluded. Marked narrowing of right circumflex and posterior descending.	No definite old infarct.
3	W. W.	M 52	Marked sclerosis of first portion of left coronary, practically complete obliteration of left circumflex. Moderate sclerosis of left anterior descending and marked sclerosis of distal 2/3 of right coronary.	Lateral portion of anter wall of left ventricle, late and posterior walls.
4	W. W.	M 60	Complete occlusion of left anterior descending and descending branches on anterior wall of left ventricle. Right coronary almost occluded 3 cm. from orifice.	Lower anterior part of l ventricle, lateral wall a lower septum. Few fibr scars in right ventricle.
5	J. S.	M 63	Narrowing of left anterior descending. Complete occlusion of right 3 cm. from origin and also at right margin of heart.	Fibrous replacement of mus in upper 1/3 of anterolate region of left ventricle.
6	A. W.	M 54	Marked calcification, but no occlusion, of left coronary espec. anterior descending branch. Probably complete closure of right circumflex at region of septum.	Old scar of anterior surface left ventricle.
7	C. H.	M 50	Both coronaries sclerotic and almost occluded in places. Left anterior descending practically completely closed. Left circumflex where it descends left lateral border freshly occluded. Right almost occluded 1 1/2 cm. from its origin and also distal to this.	Entire posterior wall of ventricle, posterior 1/2 of s tum and apex.
8	K. H.	M 55	Orifice of right coronary does not admit a probe. Right descending and left anterior descending occluded.	Upper part of posterior also upper part of ante wall of right ventricle.
9	J. L.	M 56	Complete occlusion with old and new thrombus of left circumflex. Other arteries negative.	Aneurysm of left lateral w
10	R. F.	M 64	Old calcified occlusion of a right branch of the anterior descending branch of left coronary. Moderately fresh occlusion of a left branch of the same artery.	Apex and anterior wall of ventricle.
11	H. B.	M 56	Complete occlusion of interventricular branch of left coronary.	Middle 1/3 of septal wal left ventricle.
12	D. C.	M 59	Old occlusion left anterior descending and of right coronary 1/2 cm. from its origin. Almost complete closure of left circumflex.	Apex, tip of septum, ante wall of left ventricle and a of right ventricle. Also of left ventricle, posterior lateral.
13	A. C.	M 62	Left coronary occluded by fresh thrombus 1 cm. from origin, old occlusion beyond this. Circumflex is patent but its descending branch is practically occluded. Right coronary has fresh thrombus 1 cm. from origin and entirely occluded beyond this.	0
14	S. C. H.	M 58	Luetic aortitis with complete closure of right coronary ostium and for 1 1/2 cm. beyond.	0
15	C. A.	M 29	Luetic aortitis with complete closure of right coronary ostium.	0
16	C. H.	M 51	Old occlusion of branch of left circumflex. Fresh occlusion upper 1/3 of anterior descending branch of left coronary.	Left lateral wall of left tricle.

TABLE 3

RONARY SCLEROSIS OR OCCLUSION WITH ADDITIONAL RECENT INFARCT

Location of Old Infarct	Location of Fresh Infarct	Electrocardiogram Consistent With Anterior Infarct	Electrocardiogram Consistent With Posterior Infarct	
Upper part posterior wall of left ventricle.	Apex.		+	L fr
No definite old infarct.	Apex.		+	S sl in
Lateral portion of anterior wall of left ventricle, lateral and posterior walls.	Posterior.		+	D B w 2
Lower anterior part of left ventricle, lateral wall and lower septum. Few fibrous scars in right ventricle.	Apex.		+	L
Fibrous replacement of muscle in upper 1/3 of anterolateral region of left ventricle.	Entire posterior wall of both ventricles, lateral aspect of right, lower 1/3-1/2 septum.		+	In vo
Old scar of anterior surface of left ventricle.	Fresher process involving entire diaphragmatic surface of left ventricle and septum.	?	?	A ri
Entire posterior wall of left ventricle, posterior 1/2 of septum and apex.	Fresher process at junction of anterior and left lateral surfaces of left ventricle.		+	L hi D
Upper part of posterior and also upper part of anterior wall of right ventricle.	Fresh soft areas anterior wall of right and left ventricle and posterior wall of right ventricle.		+	Id H of
Aneurysm of left lateral wall.	Left lateral wall.	?	?	R lo tr S_1 ve
Apex and anterior wall of left ventricle.	Anterior wall left ventricle	?	?	L oc fla bu ve
Middle 1/3 of septal wall of left ventricle.	Recent extension into area of old infarct.	?	?	N 9 vi la
Apex, tip of septum, anterior wall of left ventricle and apex of right ventricle. Also base of left ventricle, posterior and lateral.	Fresh infarct in left ventricle between two old areas.	?	?	In in
0	0	Neg.	Neg.	N no we at
0	0	?	?	A bl
0	0		+	In
Left lateral wall of left ventricle.	Fresh infarct of lower 2/3 anterior wall left ventricle.	+		L ch in

Comment
Low voltage and slight inversion of T_3 when fresh infarct was two weeks old.
Slurred Q-R-S with sharp inversion of T_3 and slight of T_2. Q_3 disappeared after final apical infarct.
Deep inversion of T_3 and moderate of T_2. Before death T_3 was less inverted and there was sagging of S-T interval in leads 1 and 2 and final intraventricular block.
Low voltage. Slightly inverted T_2 and T_3.
Inverted T_2 and T_3. Slurred Q-R-S. Low voltage.
Auricular flutter, low voltage and probable right bundle branch block.
Left axis deviation with diphasic T_1 and high origin and late inversion of T_2 and T_3. Deep Q_3.
Idioventricular rhythm. Left axis deviation. High ST_1, and T_2, next day high take-off of T_2 and T_3 with late inversion of T_3.
Records taken only during 9 months following original occlusion. Showed intraventricular block of unusual type with slurred S_1 and upright slurred S_3, inverted Q-R-S_3. Inverted T_2 and T_3 and at times T_1.
Left bundle branch block which after later occlusion showed diphasic T_1 and T_2 and flat T_3 and less intraventricular block. Left bundle branch block then recurred with inverted T_3.
No ecg. taken after recent occlusion. Ecgs. 9 months before death showed left axis deviation. P-R interval .22 sec. and slight late inversion of T_1.
Intraventricular block—left branch type with inverted T_1.
Normal ecg. 2 1/2 years before death and nothing but slight late inversion of T_3 3 weeks before death. No ecg. after acute final attack of pain. All coronaries occluded.
Auricular fibrillation and left bundle branch block.
Inverted T_2 and T_3.
Left bundle branch block was present but change of Q_1 and T_1 occurred with fresh infarct.

bosis at autopsy*. This group had, therefore, survived severe myocardial insults in the past but in most instances old scars were to be found which might alter the electrocardiograms resulting from the terminal acute process. Table 3 shows the location of the old and new infarcts and the character of the electrocardiograms.

of the left ventricle; in one the posterior part of the left ventricle was also involved; while in the fourth no infarction was found but luetic occlusion of the left coronary ostium. In the one case of right bundle branch block the anterior wall of the left ventricle showed old infarction but the entire diaphragmatic surface

TABLE 4

ELECTROCARDIOGRAPHIC ABNORMALITIES, EXCLUSIVE OF T WAVE CHANGES, WITH COMBINED OLD AND NEW INFARCT

Case No.	Location of old infarct	Location of fresh infarct	Partial A-V Block	Auricular Fibrillation	Auricular Flutter	Rt. Bundle Branch Block	Lt. Bundle Branch Block	Low Voltage	Idioventricular rhythm	Intravent. Block —Indefinite type	Left axis deviation
1	Post. left ventricle	Apex						+			
	Lower anterior left vent.										
4	Lateral wall. Lower septum.	Apex						+			
5	Upper 1/3 anterolateral wall left ventricle	Entire posterior both ventricles, lat. side of right, lower 1/3-1/2 septum						+		+	
6	Anterior left ventricle	Entire diaphrag. surface left ventricle and septum			+	+		+			
8	Upper part anterior and posterior right ventricle	Anterior right and left and posterior right vent.							+		
9	Left lateral	Lateral wall left vent.								+	
10	Apex and anterior left	Anterior left vent.					+				
11	Middle 1/3 septal wall left ventricle	Extension into old infarct	+								+
12	Apex, tip of septum, ant. left vent., apex rt. vent. Base post. and lat. left vent.	Between apex and base left ventricle					+				
16	Left lateral wall of lt. vent.	Lower 2/3 ant. wall left ventricle					+				
14	None	None		+			+				
			1	1	1	1	4	4	1	2	1

Table 4 shows the position of the infarcts in the heart in the cases with low voltage and disturbances of rhythm or conduction.

This table needs little comment. The only abnormalities, aside from the T wave changes shown in table 5, that occurred with any frequency were low voltage (no deflection of the Q-R-S exceeding 5 mm. in amplitude), and intraventricular conduction defects. Low voltage (four cases) appears to be found in cases with extensive infarction of the left ventricular wall. There were four cases of left bundle branch block. In two the infarction was limited to the apical, anterior and lateral portions and the septum were involved in the fresh infarct from thrombosis of the right circumflex near the septum. The two cases with intraventricular block of less definite type showed in one case extensive infarction of both ventricles and in the other a process limited to the left lateral wall of the left ventricle.

In the one case with partial A-V block the septum was infarcted.

No microscopic study of the His bundle was made in any of our cases.

Analysis of T wave changes in this group is difficult (table 5). Six cases had intraventricular block of a degree high enough to obscure the characteristic alterations in the T deflec-

*In three cases definite infarct was not demonstrable.

tions. In one of these, however, the T_3 change suggested a posterior infarct and at autopsy an old and new infarct of the lateral wall of the left ventricle was found with aneurysm of the ventricular muscle. In two others with left bundle branch block, fresh infarction of the anterior wall of the left ventricle produced in one an inversion of the T wave in lead three* and in the other a change in the Q and T waves in lead one.

Of the sixteen cases of the group with old and acute coronary narrowing and occlusion there were nine that showed T wave changes characteristic of coronary occlusion. Only one was of the T_1 type although three others had fresh occlusion in the apex of the heart and one bosed 3 cm. from its orifice. In the third instance (case 15) no infarcts were found but the right coronary ostium was occluded by a luetic process in the aorta.

Case 13 had advanced coronary disease with old and fresh occlusion of the left coronary, practical occlusion of the descending branch of the left circumflex, old occlusion of the right with fresh thrombi in both right and left. No electrocardiogram was made after the final attack of pain from which he died in twenty-four hours. No infarcts were found in the ventricular wall and the muscle was negative to microscopic examination. Electrocardiogram taken two and one-half years before death was normal as was one taken three weeks before death. His

TABLE 5

T Wave Changes in Relation to Position of Old and New Infarct from a Total Series of 16 Cases

Type	Position of Old Infarct	Position of New Infarct	Case No.	No. of Cases
T_1	Middle 1/3 septal wall of left ventricle	Same	11	1
T_3	Upper posterior left ventricle	Apex	1	
	Lateral and posterior left	Posterior	3	
	No definite old infarct	Apex	2	
	Lower anterior and lateral wall of left and lower septum	Apex	4	
	Upper 1/3 anterolateral left ventricle	Posterior left ventricle, lateral right, lower septum	5	8
	Posterior left, posterior 1/2 septum and apex	Junction of anterior and lateral surfaces of left ventricle	7	
	Upper anterior and posterior right ventricle	Anterior right and left and posterior right	8	
	No infarct but right coronary ostium completely closed	0	15	
				9

in the anterior and lateral surface of the left ventricle. The tracings in these four cases were all taken within three weeks of the fresh infarct. Reference to the table shows, however, that in the T_3 group (eight cases) a lesion was found in the posterior wall of the left ventricle in all but three cases even though in some instances a fresher lesion was found at the apex.

Explanation of the inconsistency in these three cases may be found in the individual conditions. In one (case 2) although no old infarct was found in the posterior part of the left ventricle and the anterior descending branch of the left coronary was occluded, nevertheless the left circumflex, right circumflex and posterior descending were also almost closed. In case 4 there were a few fibrous scars in the right ventricle and although the anterior descending branches of the left coronary were occluded the right coronary was almost completely thrombosed. In the third instance (case 15) no infarcts were found but the right coronary ostium was occluded by a luetic process in the aorta.

Case 13 had advanced coronary disease with old and fresh occlusion of the left coronary, practical occlusion of the descending branch of the left circumflex, old occlusion of the right with fresh thrombi in both right and left. No electrocardiogram was made after the final attack of pain from which he died in twenty-four hours. No infarcts were found in the ventricular wall and the muscle was negative to microscopic examination. Electrocardiogram taken two and one-half years before death was normal as was one taken three weeks before death. His angina pectoris had been present for only a year and a half and the findings illustrate strikingly that it is possible for occlusions of all the main coronary branches to be present without infarction of the heart wall and with normal electrocardiographic tracings.

OLD CORONARY SCLEROSIS AND OCCLUSION

Twenty-eight cases of old coronary sclerosis and occlusion with myocardial infarcts or diffuse fibrosis were found in our series. Table 6 gives the data in these cases.

It will be seen that of this group only four showed electrocardiograms characteristic of the T_1 type of change, of which one was questionable, and only four were suggestive of the T_3 change but two of these were questioned. Explanation for this probably lies in the involvement of diffuse regions of the coronary system by sclerosis, in the patchy character of the myocardial scarring and in the recanalization of older occlusions. In five cases and a possible sixth, left bundle branch block interfered with

*White, Paul D.: Electrocardiographic evidence of recent coronary thrombosis superimposed on bundle branch block resulting from previous coronary disease. Am. Heart J. 10: 260 (Dec.) 1934.

TABLE 6

OLD CORONARY SCLEROSIS OR OCCLUSION WITH MYOCARDIAL

No. of Case		Age and Sex	Coronaries Involved	Location of Infarct
1	H. B.	M 76	All sclerosed. Fresh thrombus right circumflex.	Old infarct of apex, lower septum lateral wall of left ventricle.
2	S. G.	F 70	Marked calcification all coronaries, most in left ant. desc.	Old infarct apex, adjoining septum anterior wall of left vent. with bulging.
3	J. D.	M 72	Left anterior descending.	Old aneurysm anterior left ventricle.
4	W. B.	M 70	Left anterior descending.	Anterior left ventricle and apex.
5	C. K.	M 39	First portion left coronary.	Old diffuse fibrosis of left ventricle.
6	W. Z.	M 55	Branch of left anterior descending.	Old scar mid-way anterior wall left vent
7	R. B.	F 52	Left anterior descending.	Old aneurysm of small part of left v wall near septum with perforation of sept
8	J. L.	M 62	Left anterior descending.	Old scar at apex left ventricle.
9	T. L.	M 66	Complete occlusion of right and left near origins.	Diffuse fibrosis.
10	R. P.		Marked sclerosis of many. Recanalized right circumflex.	Diffuse fibrosis.
11	F. B.	F 76	Narrowed orifices with irreg. calcification of all main branches. Practical occlusion of left ant. descending, left circumflex and right descending 2 cm. from aorta.	Diffuse old involvement of posterior wall left ventricle from septum to apex w slight bulging of anterior wall of left v tricle just above the apex.
12	W. J.	M 72	Old complete occlusion left anterior descending and right circumflex. Left circumflex practically occluded.	?
13	F. G.	M 63	Extensive atheroma of left. Old occlusion of right 2 cm. and 9 cm. from origin.	Diffuse scarring of left ventricle, n marked in region of marginal branch of circumflex.
14	G. S.	M 65	Diffuse narrowing but no occlusion.	Diffuse fibrosis.
15	C. H.	F 66	Left ant. descending almost occluded. Marked narrowing left circumflex, less of right.	Old scar of right anterior portion of ventricle, near septum and almost to apex.
16	G. W.	M 33	Sclerotic plaques but all arteries patent.	Old scar at apex with thinning of muscle
17	E. D.	M 55	Only one coronary present — the left—with right. ant. descending and left circumflex branches. No occlusion.	Old thinning of large area at apex, lo anterior wall of left ventricle and lower s tum.
18	J. D.	M 73	Narrowing but no occlusion.	Anterior and lateral parts of left ventri toward the base show thinning of the w with fibrosis of the endocardium.
19	G. G.	M 72	General calcification and old occlusion of several small branches of left ant. descending and circumflex.	Diffuse areas of fibrosis in left ventricle.
20	G. L.	M 43	Extreme sclerosis of left with recanalized thrombus of left ant. descending.	Old scar at apex and smaller one at b (? location). Luetic aorta.
21	C. L.	M 69	General narrowing and almost complete occlusion left anterior descending.	Anterior wall left ventricle near septum.
22	F. H.	M 57	Old occlusion of two branches of left ant. descending and two branches of left circumflex.	No scars demonstrated.
23	C. F.	M 55	Occlusion of branch of left circumflex leading to infarct.	Mid portion of obtuse margin of left v tricle.
24	H. P.	M 71	Calcification but no definite occlusion of left anterior descending.	Healed infarct in apex of left ventricle w thrombus over it. Aortic stenosis.
25	F. S.	F 72	Complete occlusion of left anterior descending, left circumflex and right coronary.	Slight thinning at apex of left ventri (Died in attack of prolonged pain.)
26	J. F.	M 61	Left coronary supplying apex practically occluded. Intraventricular septal branch of right completely occluded.	Adherent pericardium. Diffuse scarring left ventricle on inner surface.
27	T. F.	M 40	Thickened left anterior descending, narrow left circumflex.	Under surface of apex, near septum.
28	H. P.	F 62	Coronary ostia partly obstructed, left coronary sclerosed and fresh thrombus in left anterior descending.	No infarct.

E 6

MYOCARDIAL INFARCTS OR DIFFUSE FIBROSIS

t	Electrocardiographic Changes										
	T_1 Type	T_3 Type	Left Bundle Branch Block	Intraventricular Block	Atypical T Wave Changes	A-V Block	Low Voltage	Premature Beats	Auricular Fibrillation	Left Axis Deviation	Negative Ecg.
septum and	+?					+		+			
septum and bulging.		+									
tricle.	+			+							
x.			+			+					
tricle.	+						+				
left vent.	+					+		+		+	
of left vent. on of septum.					+						
					+						
								+			1 yr.
		+?		+			+				
erior wall of o apex with of left ven-		+									
											2 mos.
tricle, most branch of left					+	+	+				
					+		+				
rtion of left st to apex.				slight				+		+	
of muscle.										+	
apex, lower nd lower sep-			+								
left ventricle of the wall					+			+			
ventricle.										+	
one at base			+								
r septum.									+		
					+					+	
of left ven-					+				+		
entricle with osis.			? early							+	
ft ventricle. in.)		+?								+	
scarring of					+		+				
tum.			+			+					
			+								
	4	4	6	2	8	5	5	5	2	7	2

the characteristic T wave changes. No right bundle branch block was found.

T_1 Type. In the four cases of this type all had evidence of infarct at the apex of the heart or anterior wall of the left ventricle, and the anterior descending branch of the left coronary, or the first portion of the left coronary, was involved in the three characteristic cases. In the questionable case all the arteries were sclerosed and a fresh thrombus was found in the right circumflex.

T_3 Type. In the four cases showing the T_3 change the electrocardiogram did not so clearly correlate with the expected pathology. Of the two with typical T_3 change one showed marked calcification of all the coronaries, most marked in the anterior descending on the left with infarction of apex and adjoining septum with slight aneurysm of the anterior wall of the left ventricle. These findings seem definitely inconsistent with the electrocardiogram. The other case had involvement of all the main coronary branches and infarction at both apical-anterior region of the left ventricle and posterior wall of the left ventricle.

Of the non-characteristic cases one showed diffuse fibrosis of the heart with recanalized thrombus of the right circumflex, and the other showed complete occlusion of right coronary and anterior descending and circumflex on the left, the apex of the left ventricle showing some thinning.

Left bundle branch block. Of the six cases of left bundle branch block, five had infarcts at the apex of the left ventricle. The sixth had no infarct but showed a thrombus in the left anterior descending artery. This artery seemed involved particularly in all the other cases of bundle branch block except one which showed an anomaly of the coronary system, all the arteries arising from a common left coronary.

Less definite bundle branch block occurred in two cases. In both, the anterior descending branch of the left coronary and the apical region were the most prominent pathologic areas.

Indefinite T wave changes were recorded in eight cases. In this group diffuse scarring of the ventricles, involvement of the left margin of the left ventricle, absence of definite scars in spite of occluded arteries or involvement of both right and left coronaries seemed the factors accountable for most of the lack of characteristic changes.

Auriculo-ventricular block. Five cases showed conduction defect between auricles and ventricles. In all the left anterior descending artery was involved and in two the right was also occluded. Marked involvement of the septum was noted in one of these cases.

Low Voltage. In five cases low voltage of the electrocardiograms was observed, the deflection of the Q-R-S not exceeding 5 mm. in either phase in any lead. It is to be noted that old diffuse fibrosis of the ventricles, chiefly the left, was present in all. The coronary lesions were either extensive sclerosis without definite occlusion, involvement of the first portion of the left coronary (thus affecting its entire distribution), or occlusion in both coronaries.

Auricular fibrillation was present in two cases. In one almost complete closure of the left anterior descending branch with infarct of the anterior wall of the left ventricle was demonstrated, and in the other an occlusion of a branch of the left circumflex with infarct of the midportion of the obtuse margin.

Left axis deviation of considerable degree occurred in six, all with pathology in the left ventricle.

Negative Electrocardiograms. In two cases electrocardiograms were found to be essentially negative one year and two months respectively before death.

The former, a man of sixty-six, had angina pectoris for eleven years and died suddenly five days after the onset of phlebitis in one leg. No pulmonary embolus was found but complete thrombosis of the right coronary 1 cm. from its orifice and of the left coronary 2.5 cm. from the orifice. There was diffuse myocardial fibrosis.

The other patient was a man of seventy-two who died suddenly two weeks after an operation for cancer of the lip after suffering from angina for seven years with radiation of pain to the right arm. Old occlusion of the left anterior descending and right circumflex and practical occlusion of the left circumflex were found. Unfortunately no description of the condition of the muscle was given.

SUMMARY AND CONCLUSIONS

The study here presented reports the autopsy experience at the Massachusetts General Hospital for twenty years (1914-1934) in proved cases of coronary occlusion that had had electrocardiographic records shortly before death. Sixty-one cases are analyzed. Complete or recanalized thrombosis of a main artery or a main branch was found in the left coronary system in forty-six cases and in the right in twenty-one. In the same period of time 437 cases of coronary occlusion have been diagnosed clinically in this hospital.

It is clear that cases with acute coronary thrombosis with closure limited to a single coronary artery or branch of an artery are relatively rare at the autopsy table. Only seventeen such cases are so designated in our series and not all of these are completely uncomplicated. The development of the Q_1T_1 and the Q_3T_3 changes in this group is most typical and it is this change in the electrocardiogram which is specific for the localization of myocardial infarct. Inversion of the T waves alone may be seen in all leads in pericarditis, rheumatic carditis, uremia, in rare unexplained cardiac dis-

ease in which the coronary system is normal at autopsy, and apparently in relation to anoxemia and possibly coronary spasm as a temporary phenomenon.

When acute cardiac infarction is limited to the apex and anterior wall of the left ventricle our figures indicate an accuracy of seventy per cent in the localization of the infarct by the electrocardiogram from standard limb leads. In posterior or diaphragmatic infarcts or infarcts of the right ventricle the electrocardiogram was typical and diagnostic in only thirty per cent but this was influenced by the presence of intraventricular block and the series was small. It appears likely that more careful study with serial electrocardiograms and precordial leads will improve these figures although as yet the use of precordial leads appears of more value in localizing anterior rather than posterior infarcts. The occurrence of intraventricular block is a serious handicap to accurate antemortem localization of infarcts in the myocardium but in some instances repeated tracings are of value in diagnosing the advance of the occlusion or the development of a fresh one. Apparent inconsistencies between the pathologic and electrocardiographic findings should lead at necropsy to a more careful study of all parts of the myocardium even when the position of the fresh infarct seems obvious. However, high degrees of coronary thrombosis with fresh infarction can occur with normal electrocardiograms. In the three cases in our series the infarction was in the posterior wall of the left ventricle in two and diffusely over the endocardium of the left ventricle in one.

In the group of sixteen cases with old and fresh occlusion, low voltage and intraventricular block become more prominent (eleven cases), and the T wave changes less typical. In our series only one case of the T_1 type appeared and this was not entirely characteristic. Eight cases of the T_3 type were recorded but apparently there was no more relationship to the fresh infarct than to the older process and in one case no infarct was found but the right coronary ostium was closed by a luetic process in the aorta. In this group also a case was found with occlusion of all the main coronary branches without myocardial infarction with normal electrocardiogram three weeks before death.

The final group of twenty-eight, with old coronary and myocardial pathology, consists of cases many of whom were diagnosed as coronary thrombosis only at autopsy although angina pectoris was the presenting symptom in thirteen, cardiac asthma in twelve, and dyspnea on exertion in two. Congestive failure was a frequent cause of death. Only eight cases had electrocardiograms characteristic of either anterior or posterior cardiac infarct. Intraventricular block was present in eight cases and low voltage in five. T wave changes of an indefinite nature occurred in eight. It was clear by electrocardiogram that myocardial disease based on coronary arteriosclerosis was present in all but two cases and in these two the clinical history revealed prolonged angina pectoris; but little hope of localizing the cardiac infarct could be expected from a study of this group. However, had serial electrocardiograms been taken for several years before death the position of some of these infarcts might very well have been deduced. In the entire group of forty-four cases showing old coronary occlusion with or without fresh occlusion the electrocardiogram was diagnostic of cardiac infarct, irrespective of its location, in 38 per cent.

This study is intended to summarize the situation in relation to coronary thrombosis in a general hospital in which there has been an active interest in the problems of coronary disease for many years but in which no special techniques have been used for demonstrating coronary and myocardial pathology at autopsy. It illustrates what can be expected in the routine work of the electrocardiographic laboratory and autopsy room and it points the way toward more intensive study by both departments in order to utilize the material which may be available in the future for the solution of the problems of diagnosis and prognosis still remaining in this condition.

THE THYMUS SUPERSTITION*

BY HENRY W. HUDSON, JR., M.D.†

PHYSICIANS interested in the care of infants and children are confronted frequently with the question of the thymus as a possible source of danger. This may be illustrated by two recent experiences. Tonsillectomy had been recommended for a child. The mother stated that she wished an x-ray examination of the thymus, giving as her reason the statement volunteered by an osteopath friend that it was necessary and a routine in the hospital with which he was affiliated. As confirmatory evidence another friend, a roentgenologist, had stressed the importance and added that all doctors' children were x-rayed before operation. When informed, by the mother, of a previous operation under nitrous oxide the roentgenologist explained that ether anesthesia was more likely to be influenced by an abnormal thymus

*Read as a part of the program for the Clinical Congress of Surgeons.

From the Surgical Service of the Boston Children's Hospital.

†Hudson, Henry W., Jr.—Associate Surgeon, Children's Hospital. For record and address of author see "This Week's Issue," page 945.

than anesthesia by nitrous oxide. A second patient came for removal of a supraclavicular lipoma. Eight months before, an operation had been performed in his home city. The parents were well pleased with the result of operation but displeased with the surgeon. Previous to operation, the surgeon had stressed the importance of x-ray examination of the thymus but, due to some oversight, the examination was not made until after the successful completion of the operation. The examination was then said to have demonstrated the largest thymus ever seen in the —— hospital, and accordingly x-ray treatment was instituted. The father, an engineer, was unwilling to place his child under the care of the local surgeon a second time because of his supposed error of omission.

Recently a front page article in our lay press dealt with the death of a young person who had been in an accident. The article competed for prominence with the economic and political news of the day and commented "in children up to twelve years it has become routine practice to x-ray for evidence of persistent thymus because the shock of an operation may prove fatal. If the gland is evident, it can be reduced by further use of the x-ray until sufficiently small to permit operation. The thymus is unknown in adults. Children in whom the gland persists invariably die as the result of some shock, physical or mental, between the ages of four and five and twenty." Editorial comment in the *New England Journal of Medicine* properly deprecated the muddling of scientific facts common to newspapers and the unnecessary mental anguish they cause parents whose children require surgical treatment.

Several years ago, I witnessed a death during anesthesia due to the difficulties of administration caused by the nature of an injury. Because of the injury, the death came within the jurisdiction of a medical examiner. He was quoted by the newspapers to the effect that children often die under anesthesia. This was interpreted by some as indicating a death under anesthesia, due to the thymus.

Such professional experience, lay gossip, and newspaper comment is disquieting. We are in a somewhat chaotic condition due to the insistence of some hospitals that x-ray examination of the thymus be made before an operation of election while other equally prominent institutions have no such requirement.

One might adopt an attitude of compliance and have his patients x-rayed when the question is raised by the parent or when the hospital requests it. This would certainly be an easy way but, like other easy ways, might be a source of danger and, perhaps, tinged with scientific dishonesty.

The question may be easily stated. Is it necessary to radiate a thymus which has been interpreted as being enlarged by x-ray? Is it necessary to subject children who are to undergo an operation of election to x-ray examination of the thymus? If these questions can be answered in the negative, unnecessary parental anxiety, unnecessary expense and unfortunate legal complications can be avoided.

Let us assume that a child has died during anesthesia and that his physician has been summoned as defendant in a suit alleging negligence for failing to have a preoperative x-ray examination of the thymus. The fact that such examination is required in certain hospitals might readily lead to an unjust verdict. Regardless of verdict, and regardless of the statements of experts, publicity would ensue, and parents would be subjected to an anxiety which some feel is without foundation.

The second point I wish to emphasize is an economic one. Costs are not to be considered if an essential safeguard is to be sacrificed. But if there is, in fact, no such sacrifice an inquiry into costs is appropriate. Someone pays for such x-rays, whether patient, hospital, municipality, or foundation. There are, for example, some 4,000 anesthesias administered on the public service of The Children's Hospital each year. I am informed that the actual cost of a single x-ray examination is not less than $3.00. An annual expenditure of about $12,000 is of some moment. A charge of $10.00 to the private ward patient may prove a very real addition to the parent who finds the expense of a child's illness a difficult burden to bear.

One might also consider the propriety and honesty of continuing a practice for which there is little support and against which there is a mass of critically compiled data.

Although a number of authors had questioned the rôle of the thymus in sudden death from apparently trivial cause it remained for Hammar[1, 2, 3] by establishing the normal growth phenomena of the thymus, to refute the misconceptions generally held. His studies seriously questioned the theory that death under anesthesia and during operation might be caused by an enlarged thymus. In 1926 the Medical Research Council and Pathological Society of Great Britain formed a committee to collect information on a large scale, for the purpose of establishing standards and investigating closely the precise cause of death in persons dying suddenly from unexplained or trivial causes when the only apparent abnormality was the presence of a large thymus. An analysis of this data was presented in 1931 by Young and Turnbull[4]. The standards were in agreement with those of Hammar. Young and Turnbull write, "An abnormally large thymus in itself cannot be considered to be the indication of status thymicolymphaticus when no obvious cause of death is

found post-mortem'' and again, ''In the opinion of the Committee the facts elicited in the present inquiry are in harmony with those of Hammar (1926 and 1929) and Greenwood and Woods (1927) in affording no evidence that so-called status thymico-lymphaticus has any existence as a pathological entity.''

Another very careful study was made by Boyd[5] and reported in 1932. She states (referring to the x-ray studies of Liss, Wasson, Shannon, and of Cooperstock) ''These roentgen findings agree with the anatomic ones, that the largest thymuses occur in healthy children killed suddenly by accidents and that the thymus weight-body weight ratio decreases with age.'' And again, ''at all ages the variability of the weight of the thymus is high.'' Applying her findings, she states that both the mean and median weight increase rapidly in the first months of life, double their birth size by two years, and triple it by eleven years; then both decrease slowly to about birth size at senility. She also states ''when illness has lasted longer than twenty-four hours, the weight of the thymus is reduced regardless of the cause of death with the exception of tumors of the thymus, leukemia, and exophthalmic goitre. The concept of a pathological state arose from misconstruing the normal prominent thymus and lymphoid tissue for a constitutional abnormality and vice versa the involuted, inconspicuous thymus of inanition being misconstrued the normal.'' Her paper includes references to the observations of other authors.

Garland[6] examined the records of 1564 autopsies performed at the Massachusetts General Hospital. In twenty-three instances thymus glands in excess of the generally accepted normal weight were encountered. Eleven had survived the immediate effects of major operations. One case only had died during operation (sigmoidostomy for intestinal obstruction). Nine cases had had an x-ray examination, in only one of which was possible thymic enlargement suggested. Four cases might have been clinically grouped as thymic deaths, but in all four sufficient other pathological changes were found to account for death. His concluding statement is, ''The present vogue of x-raying for thymus and radiating all cases in which a shadow appears would seem to be unjustified.''

Epstein[7] reported the cause of death and incidence of disease in a series of 1000 consecutive autopsies at the Children's Memorial Hospital. Twenty-two cases were classed as status lymphaticus. Epstein states such a classification is made with reluctance but the finding of a well-nourished body, generalized lymphadenopathy, large thymus, and petechial visceral hemorrhages, taken with a history of sudden death from unexplained cause make such a diagnosis necessary. The thymus was weighed in seventeen of these cases. The largest weighed 50 Gm., the smallest 22 Gm., and the average was 32.5 Gm. In six, pneumonia was demonstrated microscopically. Whether any of these children had died during anesthesia or operation is not stated. In view of the work by Hammar, Boyd, and the Status Lymphaticus Investigation Committee, it is doubtful if these thymuses were enlarged. They may represent instances of the normal thymus and lymphoid tissue of well-nourished children who die suddenly. The fact that no other cause of death was ascertained in seventeen, does not necessarily incriminate the thymus.

Farber[8] has studied the protocols of 2000 consecutive necropsies at the Boston Children's Hospital. This covered a twelve-year period during which autopsies were obtained in seventy per cent of the deaths. About 50,000 operations under anesthesia were performed during this time. He was unable to find ''a thymus which we could regard as pathological in size, nor have we found in any case a thymus which has been associated with definite symptoms during the life of the individual.'' Farber points out the necessity for painstaking observation and thorough examination if the true cause of death is to be uncovered.

These are only a few of the papers but they are critically written after careful observations of a large amount of material. Papers expressing different views may be found but do not offer impressive evidence for the views held.

We are able to present certain information from the Children's Hospital which is in harmony with the beliefs which have been expressed. There are about 4,000 children anesthetized each year. Economy at the expense of safety has never been a custom of the hospital. It is the opinion of those responsible for policies that preoperative x-ray examination of the thymus is neither necessary nor desirable and such examinations have never been a routine. Dr. P. J. Mahoney has analyzed the data from the records of 16,195 children anesthetized between January 1, 1930, and September 1, 1934. There were 225 deaths (1.4 per cent) and autopsies were obtained in seventy per cent. He was unable to find a death which could be attributed to the thymus.

One is familiar with the customs of those making use of the private ward. There each year about one thousand children are anesthetized (in addition to the figures mentioned above). In a few instances, physicians have an x-ray examination of the thymus made before an operation of election but these are very much in the minority. An examination of the published reports of the hospital reveals no death attributed to the thymus since the private ward was opened. Apparently the experience of the private ward staff is similar to that of the full staff of the hospital.

To supplement the information I wrote to six-

teen physicians in this community. These were not selected for any personal reason, nor because their views were known, but because they have had an unusually large experience with children. Eleven replied and were willing to be quoted. Eight are in private practice and three in full-time clinical positions. Nine occupy professorial chairs. Four pediatrists, four otolaryngologists, two surgeons, and one orthopedic surgeon replied.

Of the eleven, nine had never witnessed a "thymus death", one did not answer this question and one did not answer it directly but implied that he had seen such a death.

Nine stated that they believed routine preoperative x-ray of the thymus neither necessary nor desirable, one did not answer, and one believed it to be necessary and desirable.

One very kindly inquired into 11,000 tonsillectomies in another institution and stated that in that group there had been no thymus death.

Two stated that they did not consider x-ray examination of the thymus, as ordinarily made, an accurate criterion of the size of the thymus. Two stated that if the examination was made and interpreted as showing thymic enlargement, radiation would be recommended even in the absence of symptoms. One of these added that he did not believe it was necessary but that it was in accordance with general medical belief. Three definitely stated that, in the absence of symptoms, radiation would not be recommended even though x-ray was interpreted as showing enlargement. Two stated that treatment of a structure whose function is not understood by an agent whose remote effects are poorly understood seemed to them to be potentially dangerous. This comment is noteworthy in view of the recent investigations of Rowntree, Clark and Hanson[9] which indicate that "Thymus Extract (Hanson) has accelerated the rate of growth and development, has hastened the onset of adolescence in the offspring of treated rats, and has seemed to increase the fertility of parent rats."

Dr. J. L. Morse's views have been expressed in two papers[10, 11]. He considers that "there is much doubt whether the deaths that are attributed to status lymphaticus during anesthetization and operation are really due to it. There is no proof that enlargement of the thymus is a primary or causative factor in the complex described as status lymphaticus. There is no justification, therefore, for the assumption that shrinkage of the thymus with the roentgen ray will have any effect on status lymphaticus. There is much reason to believe that many of the roentgenograms taken do not show the real size of the thymus and much evidence to show that it is very difficult to decide from a roentgenogram whether the thymus is larger than it ought to be in the given child at a given time. It does not seem either reasonable or justifiable, therefore, to say that a roentgenogram should be taken of every child before anesthetization or operation, that treatment with the roentgen ray should be given in every case before anesthetization and operation, if the roentgenologist thinks that the shadow is enlarged, and that the physician or surgeon who does not follow this course of procedure is negligent."

CONCLUSION

The evidence does not support the contention that the thymus may be a cause of sudden death in infants and children.

Unnecessary parental anxiety, unnecessary expense, and unfortunate legal complications, can be avoided.

The continuation of an unwarranted fear by physicians and hospitals is undesirable.

REFERENCES

1. Hammar, J. A.: Fifty years of thymus investigations. Ergebn. d. Anat. u. Entwcklngsgesch. 19: 1, 1909.
2. Ibid: The human thymus in health and disease. Ztschr. f. mikro. anat. Forsch. 6: 399, 1926.
3. Ibid: Die Lehre vom "Status thymicus" im Lichte der normalen Thymus Verhältnisse. Klin. Wchnschr. 8: 1385 (July 23) 1929.
4. Young, M., and Turnbull, H. M.: An analysis of the data collected by the status lymphaticus investigation committee. J. Path. & Bact. 34: 213, 1931.
5. Boyd, E.: The weight of the thymus gland in health and disease. Am. J. Dis. Child. 43: 1162 (May) 1932.
6. Garland, J.: Some side lights on the thymus vogue. New Eng. J. Med. 200: 59 (Jan. 10) 1929.
7. Epstein, I. M.: The causes of death and the incidence of disease in children. Am. J. Dis. Child. 41: 1362, 1931.
8. Farber, S.: Fulminating streptococcus infections in infancy as a cause of sudden death. New Eng. J. Med. 211: 154 (July 26) 1934.
9. Rowntree, L. G.; Clark, J. H., and Hanson, A. M.: The biologic effects of thymus extract (Hanson). J. A. M. A. 103: 1425 (Nov. 10) 1934.
10. Morse, John L.: The thymus obsession. Boston M. & S. J. 197: 1547 (Feb. 16) 1928.
11. Ibid: The thymus and status lymphaticus. J. Mich. State M. Soc. 29: 271 (April) 1930.

FRACTURES OF THE HEAD AND NECK OF RADIUS—SEPARATION OF UPPER RADIAL EPIPHYSIS

BY STEPHEN G. JONES, M.D.*

FRACTURES of the head and neck of the radius comprise 17 per cent of all elbow fractures in a group of cases treated in a seven-year period between 1923 and 1930 at the Massachusetts General Hospital. They were 1.7 per cent of the total number of fractures during the same period. They constituted the third largest group of elbow injuries. Though these figures are not startling at a casual glance, the disability resulting from this group of injuries may be very crippling. Fracture of the head and neck of the radius is a serious injury and while the prognosis is good for recovery of a useful elbow, rarely is it a normal elbow. In a comparative study with other elbow injuries, the results in this group were less good than in any other type of fracture or dislocation of the elbow. It is a fracture occurring chiefly during the second, third, and fourth decades of life. In a series of 78 cases, 35 cases occurred between the ages of 20 and 40 years, with 15 cases in the 10 to 20 age period.

Of the 78 cases, the distribution between male and female was nearly equal, there being a few more male cases. There was one more fracture of the right than the left, equally divided between males and females in respect to rights, and lefts predominating in the males. Forty-six of the 78 cases were treated in the Emergency Ward and Out-Patient Department, and comprise the simple fractures in the group. The 32 cases treated in the House were the more severe ones, or cases coming to the hospital with old injuries requiring special attention of one kind or another. End result studies have been accomplished chiefly on this group of House cases.

TABLE 1

MISCELLANEOUS DATA ON 78 CASES

	Male	44 = 56%
	Female	34 = 44%
	Right	35 = 51%
	Left	34 = 49%
		9 not stated
Male	Right	= 18 = 51%
	Left	= 21 = 62%
Female	Right	= 17 = 49%
	Left	= 13 = 38%
46 cases treated in E.W. and O.P.D.		= 59%
32 cases treated in House		= 41%

The problem involved in a fracture of the head or the neck is the same, the disability resulting is similar, so no effort is made in this series to distinguish between them. Anatomically, the head consists of the imperfectly circular portion with the depression on the top or articular surface which articulates with the capitellum, and an edge which is in relationship with the lesser sigmoid cavity of the ulna. Its anterior posterior diameter is the greater. The neck is a constricted cylindrical portion about 1 inch in length. The epiphysis is one of the latest of the long bones to unite to the shaft, occurring at the age of 14 to 15 years. Below the age of 7, the epiphysis is largely cartilaginous and able to resist injury, so that the age period in which epiphyseal fracture may be produced is relatively short. The osseous center represents only a thin disk of bone and corresponds to the radial head. The orbicular ligament which attaches to the inner side of the head and neck makes them intra-articular, but is of very little importance in the mechanism of fracture except in limiting rotation.

TABLE 2

LIST OF COMPLICATING INJURIES

78 Cases

30 Cases with Complicating Fractures = 38 Per Cent	
Posterior-medial Dislocation of the Elbow (with fracture of coronoid process 7)	10
Fracture of Olecranon Process	8
Fracture Upper Third of Ulna into Elbow Joint	2
Intercondylar Fracture Lower End of Humerus	7
Os Calcis	1
Colles	1
Patella	1
	30

Injuries of the upper extremity of the radius may be produced by indirect violence, the force being transmitted along the shaft to the radius, driving the head against the capitellum. The patient falls upon the pronated hand with the arm extended. More rarely direct violence is the cause, acting on the lateral aspect of the upper forearm or elbow. It is sometimes stated that torsion is a causative factor, but since the possibility of this has been disputed by dependable authorities, it remains a mute point; it is adequate simply to mention it in passing. Whatever the mechanism, in a fairly high proportion of cases (38 per cent in our series) the fracture occurs in association with some other bony injury, particularly posterior-medial dislocation of the elbow with fracture of the coronoid process (7), fracture of the olecranon process (8), frac-

*Jones, Stephen G.—Surgeon to Fracture Clinic, Massachusetts General Hospital. For record and address of author see "This Week's Issue," page 945.

ture of the upper third of the ulna into the elbow joint (2), and intercondylar fracture of the lower end of the humerus (7).

The common types of fracture occur. It may be a complete fracture, incomplete, comminuted, impacted, partial, or the whole head may be displaced off the shaft. The symptoms are those of fracture elsewhere: pain, tenderness, swelling over the outer aspect of the elbow. The arm is held in semiflexion and pronation. Supination is limited. There is loss of function. Crepitus is rare. Fragments may be displaced out, in, or down. The diagnosis should be confirmed by x-ray.

SEPARATION OF THE UPPER RADIAL EPIPHYSIS

The epiphyseal fractures numbered 3, or 9 per cent, in our group of 32 House patients. patients in that age range with but 3 separated epiphyses or 15 per cent. Summaries of the records of the patients with epiphyseal fracture are presented in table 3.

FRACTURES OF THE HEAD AND NECK OF RADIUS

Fractures of the head and neck of the radius, when accompanied by displacement, commonly cause serious impairment of elbow joint function. Not only does the bony deformity limit or prevent rotation of the upper articular element in the lesser sigmoid cavity of the ulna with loss of the ability to pronate and supinate the forearm, but there is frequently limitation of flexion and extension of the elbow as well. This is due to scar formation and thickening in the anterior capsular ligament, usually the result of the displacement of a fragment of bone in this direction.

TABLE 3

Identification	Age	Age of Injury	Complications	Displacement	Treatment	Result	1 year
R. E. W.266360	13	2 days	Fracture of olecranon without displacement.	Marked +++	Open reduction performed 5 days after injury. No internal fixation. Elbow splinted in acute flexion.	Good	Epiphysis closed.
N. K. W.276613	8	6 days	Fracture of olecranon without displacement. (Feeble-minded.)	Marked +++	Elbow fixed in sling at right angle flexion. Reduction not attempted.	Fair	Supination lacks 15°; extension lacks 20°. Loose body in joint.
L. P. O.276718	8	14 days	Greenstick fracture of ulna.	Marked +++ Callus present.	Open reduction performed. Fragment loosened and replaced. Held in apposition by fixation of elbow in position of acute flexion.	Good	Only about 20° rotation of forearm. Flexion and extension normal. Epiphysis closed.

Two of the patients were aged 8 years, and one, 13 years. This conforms with the dictum that this injury occurs only within the ages 5 to 17 years. When the epiphysis is fractured and displaced, it is apt to become isolated as a loose body in the joint, devoid of blood supply. This occurred in one of our 3 patients when no operation was performed to replace the epiphysis (a feeble-minded patient). In the other 2 patients, open reduction and replacement were performed, and union in good position was obtained. No method of internal fixation was employed, the fragment being retained in alignment by the position of acute flexion of the elbow. Obliteration of the epiphyseal cartilage occurred in both cases, but no appreciable shortening of the radius resulted since the amount of growth contributed by the upper epiphysis at this age is slight.

It does not follow that patients injuring the upper extremity of the radius in the age limit 5 to 17 years all separate the upper radial epiphysis. In our series of 78 cases, there were 20

Ossifying hematoma or myositis may also cause marked limitation of motion. This is particularly true in the case of fractures associated with dislocation of the elbow. It also may follow resection of the radial head, but is not necessarily the result of operation. To guard against this complication at the time of operation, it is necessary to obtain careful hemostasis and to avoid leaving any loose bone fragments. This is a common error.

To prevent future disability, it is necessary to correct the bony deformity, but the situation and nature of the fracture are often such as to render it impossible except by open operation. This may take the form of open reduction with replacement of fragments, removal of fragments, or removal of the entire radial head. When the bony displacement is absent or slight, the treatment indicated is a protective Lund swathe or sling for about three weeks, with gentle mobilization started the fifth to seventh day following injury. In operative cases, more stable immobilization is at times desired. The

elbow is splinted in the position of right-angled flexion in a light moulded posterior plaster splint for the period of one week, following which mobilization should be begun. The types of injury and incidence in 32 House patients are given in table 4.

TABLE 4

THE TYPES OF INJURY AND INCIDENCE IN 32 PATIENTS

Epiphyseal Fractures, Upper End of Radius	3
Fissure Fractures of Radial Head without Displacement	3
Fractures of Radial Head with Displacement	15
Fractures of Radial Neck with Displacement	4
Comminuted Fractures Involving both Head and Neck	7
	32

In our group of 32 House patients with fracture of the head and neck of the radius, operative treatment was employed in 20 and consisted of open reduction in 6, excision of loose fragments in 5, and resection of the head in 9.

TABLE 5

OPERATIVE CASES OUT OF 32 HOUSE PATIENTS

Removal of Head	9 = 28%
Removal of Fragments	5 = 16%
Open Reduction	6 = 19%
	20 = 63%

The results in these were rated as good in 10, fair in 4, poor in 4, and unknown in 2.

TABLE 6

THE RESULTS OBTAINED IN FRACTURE OF THE HEAD AND NECK OF RADIUS

		Total	Good	Fair	Poor	Unknown
No Reduction or Operation Performed		12	3	2	2	5
Open Reduction and Replacement	Early	5	2	—	1	2
	Late	1	—	1	—	—
Excision of Fragments	Early	4	2	1	1	—
	Late	1	1	—	—	—
Excision of Head	Early	5	4	1	—	—
	Late	4	1	1	2	—
		32	13	6	6	7

It would seem from this analysis, with only 3 good results out of 12 non-operative cases, that more of this group should have been operated on. The 3 cases obtaining good results were treated immediately following the injury. The others were treated immediately except for 3 cases which were treated 1 day, 6 days, and 8 months after injury. Open reduction and replacement gave good results only in the 2 cases of separation of the epiphyses. Excision of fragments gave good results in certain cases. Excision of loose bone fragments should be performed only in the case of fractures involving the radial head when there is a single fragment and when at least two-thirds of the circumference of the head remains intact, including the inner half that articulates with the ulna. Excision of the head gave satisfactory results in every case performed early, but the late excisions were not so encouraging. The policy of waiting on comminuted and displaced fractures of the head and neck of the radius to see what result is obtained, with the thought that if it is not satisfactory, open operation can be performed, should be abandoned. Early operation is imperative if operation is indicated. The average hospitalization was 20 days. The period of disability ranged from 1 to 22 months, the average being 9 months to 1 year before return to work. Both the average hospitalization and disability figures are considerably influenced by the complicating injuries which were present in 38 per cent of the cases.

There are two operative approaches to the head of the radius.

The Anterior Approach. An anterior incision is made over the head of the radius. The supinator longus muscle is retracted outward exposing the musculospiral nerve. This, in turn, is retracted outward. The capsule lies directly beneath, over the head of the radius.

The Posterior Approach. A posterior lateral incision is made, the skin flap being turned upward. The musculospiral nerve is identified passing around the head of the radius. This is retracted downward with some of the fibers of the anconeus muscle, exposing the head of the radius.

The anterior approach is employed for replacing dislocation of the head of the radius. The posterior approach is preferable for other conditions of the head of the radius requiring surgery.

Conclusions:

1. Open reduction with replacement ought not to be attempted except in the case of epiphyseal fractures and occasionally in fractures of the radial neck.

2. Excision of fragments gave good results in certain cases where a single small fragment, which did not articulate with the ulna, was removed.

3. In all comminuted and displaced fractures of the head and neck, resection of the head should be advised.

4. The resection of the head should be performed early (within the first 2 weeks) rather than late.

5. Ossifying hematoma or myositis is a common complication.

6. Fracture of the head or neck of the radius with displacement is a serious injury. While the prognosis is good for recovery of a useful elbow, rarely is it a normal elbow.

REFERENCES

1. Eliason, Eldridge L.: Fractures of the Humerus, Radius, and Ulna.
2. Nelson Loose-Leaf Living Surgery. Volume III.
3. Wilson, P. D., and Cochrane, W.: Fractures and Dislocations. Philadelphia: J. B. Lippincott Company. 1928.

The Massachusetts Medical Society

PROCEEDINGS OF THE COUNCIL

Special Meeting, April 3, 1935

A SPECIAL MEETING of the Council of the Massachusetts Medical Society was held in John Ware Hall, Boston Medical Library, 8 Fenway, Boston, on Wednesday, April 3, 1935, at 12 o'clock, noon, the President of the Society, Dr. William H. Robey, in the chair and Dr. A. S. Begg, Norfolk, serving as Acting Secretary. The following 131 Councilors were present:

BARNSTABLE
Samuel M. Beale, Jr.

BRISTOL NORTH
William H. Allen
Arthur R. Crandell
B. M. Latham

BRISTOL SOUTH
George W. Blood
R. B. Butler
Philemon E. Truesdale

ESSEX NORTH
E. S. Bagnall
R. V. Baketel
J. Forrest Burnham
Henry F. Dearborn
Arthur P. George
Thomas R. Healy
E. P. Laskey
Leroy T. Stokes
W. D. Walker

ESSEX SOUTH
J. F. Donaldson
William T. Hopkins
J. F. Jordan
Walter G. Phippen

FRANKLIN
H. M. Kemp

HAMPDEN
John M. Birnie
W. A. R. Chapin
Frederic Hagler
Allen G. Rice
George L. Schadt

MIDDLESEX EAST
J. Harper Blaisdell
Richard Dutton
Joseph H. Fay
Edward M. Halligan
K. L. Maclachlan
R. R. Stratton
E. E. Tyzzer

MIDDLESEX NORTH
Archibald R. Gardner
G. A. Leahey
T. A. Stamas
Michael A. Tighe

MIDDLESEX SOUTH
Charles F. Atwood
Elmer W. Barron
C. F. K. Bean
Enos H. Bigelow
Allen H. Blake
George F. H. Bowers
A. C. Cummings
D. F. Cummings
A. W. Dudley
Wilfred G. Grandison
Fred A. Higginbotham
Norman M. Hunter
C. M. Hutchinson
Josephine D. Kable
Alexander A. Levi
L. W. McGuire
John A. McLean
Edward Mellus
Charles E. Mongan
W. D. Reid
Sumner H. Remick
C. H. Staples
H. P. Stevens
F. Van Nüys
W. S. Whittemore

NORFOLK
F. G. Balch
A. S. Begg
Horace K. Boutwell
I. A. Finkelstein
Carleton S. Francis
Alice M. Gray
Walter A. Griffin
J. B. Hall
Charles J. Kickham
Walter A. Lane
J. S. H. Leard
J. S. May
Francis P. McCarthy
Sylvester F. McKeen
H. Morrison
Benjamin Parvey
H. F. R. Watts
G. L. Gately
Robert B. Greenough
John Homans
H. T. Hutchins
Roger I. Lee
Francis W. Palfrey
W. S. Parker
G. P. Reynolds
William H. Robey
G. C. Shattuck
Warren R. Sisson
Louisa Paine Tingley
Harvey P. Towle
S. Warren
F. A. Washburn
Conrad Wesselhoeft

NORFOLK SOUTH
Charles S. Adams
William G. Curtis
George V. Higgins

PLYMOUTH
Leon A. Alley
Thomas H. McCarthy
J. J. McNamara
George A. Moore

SUFFOLK
A. E. Austin
Gerald Blake
W. B. Breed
Charles S. Butler
David Cheever
R. C. Cochrane
Fletcher H. Colby
William P. Cross
Lincoln Davis
R. Fitz
C. Frothingham

WORCESTER
J. C. Austin
Walter P. Bowers
Philip H. Cook
George A. Dix
George E. Emery
David Harrower
E. L. Hunt
Edwin R. Leib
A. W. Marsh
J. W. O'Connor
Edward H. Trowbridge
F. H. Washburn
Royal P. Watkins
S. B. Woodward

WORCESTER NORTH
Thomas R. Donovan
Francis M. McMurray
H. R. Nye
Walter F. Sawyer

The meeting was called to order by President Robey, who asked for the reading of a summary of the minutes of the last meeting. The Acting Secretary stated that the records of the last meeting were then in press and read an abstract. Since there appeared to be no errors or corrections noted by the Council the chair declared the records approved.

President Robey then stated that one of the chief objects of the meeting was to consider the report of the House of Delegates of the American Medical Association made at its Special Session in Chicago on February 15 and 16, 1935. He stated that Dr. Cody had prepared a report

of that meeting. Since Dr. Cody was not able to be present, the Secretary read Dr. Cody's report.

AMERICAN MEDICAL ASSOCIATION
HOUSE OF DELEGATES
Special Session
February 15, 1935

In compliance with the request of the Board of Trustees the House of Delegates was officially called by the Speaker to convene in Chicago on February 15 and to remain in session, recessing from day to day until its deliberations were concluded.

The business would be limited to the consideration of the social and economic policies of the Association as related to pending and proposed legislation, to sickness insurance and to other matters which might be submitted by the Board of Trustees.

One hundred and sixty-eight delegates were present when the House was called to order. Five were present from this Society.

The Board of Trustees through the Chairman, Dr. Upham of Ohio, presented a statement concerning the existing situation. Six questions were submitted for consideration. The discussion by a large number of delegates followed. When all had been heard the speaker appointed a committee of seven to which the statement of the Board of Trustees, the questions suggested for discussion and the suggestions and motions offered by individual delegates were referred. The committee went into session, heard several delegates and on the following morning submitted a report to the House. After discussion this report was referred back to the committee when amendments were made in accordance with motions by the House. On the afternoon of this day the report of the reference committee was again read and adopted by the House without dissent.

This report has appeared in the columns of our journal and the current issue of the *American Medical Bulletin*.

The minutes of the Special Session were published in the *Journal of the American Medical Association* of March 2. With such wide publicity it is omitted here.

For the Delegates,
Respectfully submitted,
EDMOND F. CODY.

Dr. Charles E. Mongan of Middlesex South moved the adoption of the report of our delegates to the House of Delegates' Special Meeting February 15 and 16, 1935 as read by the Secretary. The motion was seconded. There appeared to be no discussion and when put to a vote, the motion was carried.

Dr. Robey then stated that the next item of business was a resolution to be offered by Dr. Tighe. Dr. Michael A. Tighe of Middlesex North stated that the Public Relations Committee, at its meeting the week previous, considered the matter of the resolution adopted by the American Medical Association on February 15 and 16 last and voted unanimously to approve the action of the House of Delegates and to recommend that this Council do likewise. He stated, "You have approved the action of your House of Delegates, and this was an essential step to whatever program the Public Relations Committee might follow." He stated that the Committee wished to serve the Society and to do so it must know the Society's wishes and attitude toward certain principles. He stated, "In the event that this Council saw fit to adopt and ratify, as it has this morning, the resolution passed in Chicago, the Public Relations Committee had formulated a plan whereby we may do our part here in Massachusetts to support the stand of the American Medical Association, and, with your permission, Mr. President and Councilors, I should like to read the following:

"'The Public Relations Committee of the Massachusetts Medical Society, being aware that there is a well organized effort favorably to influence public opinion through the instrumentality of newspapers, popular magazines, and the radio in the matter of compulsory sickness insurance, and being aware that such compulsory sickness insurance is not in the interests of public good, urges the Council of the Massachusetts Medical Society to approve a plan whereby the evils of compulsory sickness insurance may be brought to the attention of the public. This plan seeks to disseminate knowledge of these evils through the press, through talks over the radio, talks to luncheon groups, service clubs, women's clubs, men's clubs, labor and sociopolitical organizations. This plan will also acquaint the public with the studies which the Massachusetts Medical Society, through its Public Relations Committee, has entered into relating to the adequacy of medical care in Massachusetts. This plan also indicates that, if this survey shows the care to be inadequate, the Massachusetts Medical Society will formulate, offer, and sponsor a remedy.'

"Mr. President, I move the adoption of the recommendation of the Public Relations Committee."

The motion was seconded by Dr. Mongan. In discussion, Dr. Mongan added to Dr. Cody's report. He stated that the attendance at the House of Delegates, at the recent meeting, was the largest in the history of the organization. The representation from all the states was full as was also the representation from the sections. Such state presidents or secretaries as were present in Chicago were likewise invited, as were representatives from the Army, Navy and the Public Health Service. The discussions were conducted with a great deal of earnestness, and much interest was manifest. When the report was brought in and was discussed very thoroughly the amendments and suggestions were accepted in good spirit by the reference committee and the amended report was finally adopted unanimously. Dr. Mongan stated that he believed the unanimity of opinion expressed in the national organization should carry great weight with our own state organization. Dr. Mongan denied that the position of the medical profession in "progressive legislation" had been a

negative one. He alluded to a number of positive contributions in the past. He believes that now we have come face to face with a medical crisis and that it is one of the concerns of the medical profession in Massachusetts. He said, "The President and his Economic Security Council recognize that fact when they say they will give you a law and they will supervise the law but each state must make its own laws, and I suppose the states are to make their own laws under the conditions such as they find in their states."

He stated that shortly there will be reported in Congress a skeleton of the President's economic security plan but for the present this will cover only old age pensions, unemployment insurance, with perhaps something on public health, and something on the care of maternity cases and crippled children. The scheme will carry with it payroll contributions and will not become active until 1937. He stated that the national government will not distribute funds under any of these laws until 1942. The crisis before us is not concerned with the national government activities. Next he referred to the compulsory insurance bill. He said that labor did not seem to be very warm on the subject and if the medical profession was willing the representative of labor stated that he would ask the legislative committee to recommend the creation of a commission to study the whole subject in Massachusetts. Therefore, he believes that our concern will be to make proper representations to the commission when it is appointed.

Dr. Mongan believes that adequate education of doctors and of the public must be undertaken in the near future. He believes that Dr. Tighe's motion offers a way to bring this about. He also referred to the survey now being conducted by the Public Relations Committee to determine the adequacy of medical care available to the citizens of Massachusetts. He believes that the results of this study and the educational campaign contemplated by Dr. Tighe's motion can reach not only the doctors but all kinds of organizations and service clubs and can present the matter impartially. The coöperation of the medical profession is essential. He pointed out the advantages for such a movement in this State because of the compactness of our organization and the high standard of medical practice which obtains here. He stated that he thought it was the duty of the profession to assist in the proper presentation of the subject matter to the public and that if this was properly done, we could expect the proper kind of legislation which would preserve not only the rights of the profession but the rights of every individual and give a medical service which would be fitting to the Commonwealth.

In response to a request from Dr. David Cheever of Suffolk the motion was reread. In the discussion Dr. John Homans of Suffolk raised a question as to the wording of the resolution which to him did not sound impartial. He stated, however, that he approved of the ideas involved.

Dr. Tighe then spoke further in explanation of the wording of the resolution and pointed out the necessity for local action by the states under the resolutions adopted by the House of Delegates at its recent meeting.

Dr. Philemon E. Truesdale of Bristol South felt that the resolution expressed a desire and an intention on the part of the medical profession to gain the confidence of the public and to retain the responsibility for the care of the sick by the medical profession and not by other agencies.

Dr. Francis P. McCarthy of Norfolk asked for further information regarding the compulsory sickness insurance bill which had been proposed in the present Legislature.

Dr. Mongan answered Dr. McCarthy's questions at some length. In response to a second question regarding national legislation, Dr. Mongan quoted from an article in the magazine *Fortune* for March, and further from a current newspaper. The quotation was as follows:

"Estimates by Secretary Morganthau indicate that aggregate collections during the first year of the tax would amount to $622,000,000. Collections would reach $18,683,000,000 in 1950, when the maximum tax would be in effect, and would continue piling up until a peak reserve of $50,093,000,000 would be reached in 1980, under the bill as now drafted."

Dr. George L. Schadt of Hampden asked if there was anything in the proposed bills that makes it mandatory and compulsory for the medical profession to serve the insurance boards. At the President's request Dr. Mongan replied that there was no compulsion but that since many physicians are feeling the pinch of the times, they are willing to do most anything and he mentioned a society recently organized in Brooklyn and now including six hundred doctors which is definitely out for the socialization of medicine.

Dr. Walter A. Lane of Norfolk raised a question regarding the program contemplated by Dr. Tighe's motion. This referred to the sponsoring of the radio broadcasting and the newspaper articles. He felt that the responsibility should be definitely placed by the Council so that there can be no question of a lack of authority by which the work could be accomplished. Dr. Lane also questioned the advisability of taking quite so broad a stand, as has been offered, without further discussion.

Dr. J. Forrest Burnham of Essex North attempted to clear the atmosphere by further explanation of the probable attitude of the administration in Washington which he believes

to be to delay any action on sickness insurance to a later date.

Dr. Robert B. Greenough of Suffolk, a member of the Medical Advisory Council to the Secretary of Labor in connection with President Roosevelt's Committee on Economic Security, spoke informally about the activities of the Medical Advisory Council. He stated that the members of this Council were called as individuals and not as representatives of different organizations. The first meeting was held in Washington in November at which time the situation was explained to them. It appears that in July, 1934, President Roosevelt appointed a Cabinet Committee on Economic Security and authorized this Committee to expend Federal funds and to organize a technical committee. This technical committee was divided into a number of sections, one to study old age pensions, one unemployment insurance, one public health service and finally one group to study health insurance. At the first meeting, the technical committee to study health insurance submitted for discussion a general list of statements concerning health insurance. These statements were discussed by the Advisory Council. The purpose of the Advisory Council was to act as advisers to the technical committee in connection with the plans which the said technical committee had been asked to prepare and submit to the Cabinet. The Council was disbanded after two days of continuous session but met again either in the latter part of January or the early part of February. At this meeting a somewhat more definite plan was submitted to the Advisory Council and again items in it were discussed. It should be understood that at no time was there a vote taken by the Advisory Council. The opinions of individuals were recorded and it was understood that these opinions were to be given consideration by the technical committee in the preparation of its report to the Cabinet Committee on Economic Security. The Council was again dismissed and it was intimated that they would probably not be called together again. The members of this Advisory Council were not at liberty to discuss or to reveal the matters which came up for discussion since all publicity was to come from higher authority in order to prevent confusion and misunderstandings. Dr. Greenough stated that he believed that the Committee on Economic Security has received a first draft of the report of the technical committee which considered health insurance, that this has been studied and that the legal advisers of the Committee on Economic Security have been asked to put the material into form so that it may be presented for legislative action after such a decision is finally reached. According to Dr. Greenough, the rather widely expressed opinion in Washington was, as Dr. Mongan intimated, that no action will be taken this year and, since the plan is said to involve action by the individual states and since only eight Legislatures meet next year, it is improbable that there will be any action in Washington in the near future. Dr. Greenough feels that the opinions expressed to the technical committee by the Advisory Council were helpful in eliminating some of the possible dangers generally recognized to exist in health insurance as it has been introduced in other countries. Dr. Greenough expressed the hope that, whether or not the matter is presented for legislative action by Congress, the form of the report of the technical committee to the Committee on Economic Security may be published, since in his opinion this action would do much to set our minds at rest. Dr. Greenough then commented upon the motion submitted by the Committee on Public Relations stating that there might be some fallacy in Dr. Homans' deduction concerning the wording of the motion. Yet he believes the wording of the motion would be strengthened by stating that the Committee feels that a thorough investigation is needed and that when and if a lack of adequate medical facilities is found to exist in Massachusetts, the Committee will be prepared to consider all possibilities and to make recommendations to the Massachusetts Medical Society as to the best procedure. Dr. Greenough's remarks were closely followed by the Council and received considerable applause.

In response to a question by President Robey for closing remarks, Dr. Tighe stated that the Public Relations Committee, after thorough consideration, has prepared a plan to deal with the situation. Such a plan cannot be entirely definite until the survey is completed. He stated that the publicity should be sponsored by the Massachusetts Medical Society. That is why the general plan is presented. He stated that the Committee on Public Relations was aware of the delicacy of this phase of work and that he was confident that before the final plans would be put into operation, care would be taken to avoid any infringement of the code of ethics. He stated that, while the idea of a subcommittee on social legislation and insurance had been in his mind from the beginning, the importance of creating such a committee was such that it should be approached with the utmost caution and should be done in close coöperation with the Committee on Ethics and Discipline. Dr. Robey then put the question and the motion was carried without dissent.

Dr. William M. Shedden of Suffolk then presented the report of the Committee of Arrangements. (See Appendix No. 1.)

Dr. Elmer S. Bagnall of Essex North commented upon the report which he believed to forecast one of the most interesting meetings that the Society has held. He felt, however, that it might

be improved if, at the Annual Meeting of the Society, provision were made for adequate discussion of medical economics. Dr. Bagnall moved that the Committee of Arrangements be requested to consider the possibility of including hereafter in the program of the Annual Meeting some discussion of medical economics.

Dr. Robey stated that Dr. Douglass V. Brown, Assistant Professor of Medical Economics at the Harvard Medical School, had recently consented to speak at the Annual Dinner on the subject of medical economics. He then asked Dr. Bagnall concerning his motion and Dr. Bagnall replied that he did not believe it feasible to include the subject in the program for this year, but he believed that it was of such vital interest that it might be included in the future and that outside speakers might be available. He mentioned the fact that he did not recall having seen the President of the American Medical Association at our meetings and it seemed to him that the presence of this official or one of his representatives might be a proper part of our program.

Dr. Robey stated that the President-Elect of the American Medical Association attended the Annual Meeting two years ago.

Dr. Burnham then seconded the motion. When the vote was taken, the chair was at first in doubt but upon asking for a standing vote this doubt was removed. The motion was carried by approximately 77 ayes and 12 nays. The chair then declared that the Council orders that Dr. Shedden's report be received.

The President called for a report of the Committee on Permanent Home stating that because of the death of Dr. Thomas J. O'Brien this Committee has been reorganized and that it is now composed of Dr. Robert B. Greenough, Chairman, and Drs. Samuel B. Woodward, Charles G. Mixter, John M. Birnie and Charles S. Butler. Dr. Greenough read the report which was signed by Dr. Mixter. (See Appendix No. 2.) The Council voted to accept the report.

The chair announced that Dr. Reginald Fitz had been appointed to serve as delegate to the American Medical Association in place of Dr. Charles H. Lawrence, who was unable to serve. Confirmation of this appointment was voted.

The President then asked for incidental business and Dr. Greenough stated that by reason of the loss to the Society of Drs. Burrage and O'Brien it seems necessary that there should be a study of the ways and means by which the affairs of the Society may be carried on efficiently and also that certain functions of the *Journal* might be considered in this connection. He stated that recommendations will have to be submitted to the Annual Meeting of the Society and that matters of such importance should receive the attention of the Fellows. Efforts should be made, therefore, to secure as large an attendance as possible at the Annual Meeting. He then presented the following motion:

"That a committee be appointed by the President which will give due consideration to any desirable changes in the administration of the affairs of the Society and prepare recommendations for action by the Council and the Society." The motion was duly seconded.

Dr. William B. Breed of Suffolk discussed the need for a closer unity between the officers of the *Journal* and the officers of the Society. He felt that this need is even more apparent from the discussions that have been held to-day. He believes that there are many difficulties in the way of the profession and he spoke of the necessity for coöperation between the Secretary and the other officers, and the *Journal* in the matter of policies for publicity. He stated that he had no specific plan to offer but recommended that the committee seriously consider some form of unification of the three departments of the Society. He suggested certain possible combinations of physicians and laymen in order to bring about continuity and at the same time insure proper carrying out of the business and executive duties which fall upon the headquarters' office.

Dr. Robey then put the question before the Council for a vote and it was unanimously carried. The committee appointed was announced as follows: Dr. Roger I. Lee, Chairman, Drs. Robert B. Greenough, Walter G. Phippen, Royal P. Watkins and George S. Reynolds.

Dr. Franklin G. Balch of Norfolk next spoke on matters which concern the Committee on Malpractice Defense. He stated that a law had been passed by the present Legislature about which he knew nothing until two days ago so that the remarks were to be interpreted as being his opinions and not of the Committee on Malpractice Defense. He stated that cases which were brought before the Superior Court may remain there for years before they are brought to trial, that in Boston it might be as long as six years. The following is the new chapter which embodies this change:

"In all actions of contract or tort for malpractice, error or mistake against physicians, surgeons, dentists, optometrists, hospitals or sanitaria, pending in the Superior Court, now or at any time, the court shall at the request of any party to the proceeding, advance the same so that it may be heard and determined with as little delay as possible."

Inasmuch as these malpractice suits could be reached under the new law in perhaps three or four months he felt that this would be of great advantage to the medical profession. He stated that there was another item attached to the bill, namely, a change in the statute of limitations but this was not allowed by the Legisla-

ture although it came very close to passing. He is of the opinion that this added item would be of great advantage to the profession and that its proponents feel that it may even yet be brought out and passed if it has the support of the Massachusetts Medical Society. He suggests that Dr. Robey as President might address a letter to the Speaker of the House expressing the opinion of the Society and that this would have a favorable result.

Dr. Robey stated that naturally he would not send such a letter without the consent of the Council.

Dr. Balch then moved that the President prepare and sign a letter similar to the one now submitted by Dr. Balch. The letter was read and the motion was seconded. (See Appendix No. 3.)

Dr. F. P. McCarthy stated that this was an important matter and that it might appear to the public that the profession was attempting to be placed in rather an exclusive position. He stated that, while in his opinion a statute of limitations of two years was too long, he would much prefer to have the Council let the matter rest and leave the law as it now stands and perhaps at some future date make some effort to compel patients to put up a bond in order to prevent spurious suits. In such an event it would appear that the profession is perfectly willing to allow a two-year period in which to bring legitimate claims.

Dr. George A. Moore of Plymouth suggested that hospitals might well be included in the list along with physicians and dentists.

Dr. J. Harper Blaisdell of Middlesex East stated that the two-year limitation or one-year limitation is a delusion and a snare. He stated that any smart lawyer can sue a physician as had happened in his case for alleged malpractice twelve years ago. It can be gotten around very simply. The patient states that ten years ago or inside the two-year limit the physician did an unlawful act and that he was not informed of this act which resulted in injury and therefore fraud was committed. It would appear, therefore, that any physician can be sued for something which occurred practically any time in the past so that as a practical matter it makes very little difference whether the limitation is one or two years.

Dr. F. P. McCarthy stated that he knew that even if an x-ray burn developed in two years or the recognition developed two or three years after the treatment, the two-year limitation does not eliminate the suit. He also stated that if a sponge is left in the abdomen and is not discovered for two or three years, suit is not prevented. He also stated that if the case were a child, suit might even be brought twenty-one years later.

The question was then asked for and upon a standing vote it appeared that the motion had been adopted.

Dr. W. A. R. Chapin of Hampden next spoke on the matter of the activities of the physicians of the State in legislative matters. He felt that the legislative program had not received proper support from various district societies. He stated that in his district the physicians had taken the matter seriously and that leading men such as Dr. Sweet had written many letters to leaders in industry, etc., asking for support of House bill 756. There was another bill in the Legislature which might serve to indicate the attitude of legislators toward this bill and upon this other measure he stated that, out of fifteen legislators in his district, thirteen voted against the bill. He stated that he understood that some other districts had done excellent work but he felt that some districts had been rather lax. He did not feel that it was fair that this should be so. He believed that when the Council voted to take a stand in any legislative matter, all districts should make every effort to support it. He believes that the influence of the physicians is an important one and, if it is properly exercised, much can be accomplished.

Dr. F. P. McCarthy referred to the meeting of the Council in Worcester at which time a proposal was made to enlarge the Legislative Committee so that each section would have representatives and be responsible to the local sections. He stated that the remarks of the last speaker emphasize the value of some such procedure and expressed the hope that when the committee, which has been appointed to-day, considers the question of legislation it will give due regard to this phase.

Dr. Samuel B. Woodward of Worcester: "Mr. President, I simply want to say that the speech from the western part of the State warms the cockles of my heart. For nearly twenty years I have almost every year tried to induce the members of this Society to support a certain bill—without success!"

Dr. Tighe spoke of the activities of Middlesex North and told how their Legislators had been entertained at dinner and how the bills were discussed. He stated that he believed the Legislators were glad to meet with the physicians and that they had expressed their thanks for the help which the district offered them in settling some of their legislative problems.

Dr. Robey stated that he was glad Dr. Chapin had brought the matter up and that, from the Society's office and from the committee on legislative matters, an attempt had been made to interest all of the members of the Society. He expressed the belief that the work this year had been fairly successful. He stated that he would

not discuss House Bill 756 upon which a great deal of work had been done but that he would make a report on this at the Annual Meeting.

Dr. Edward H. Trowbridge of Worcester presented some suggestions and moved that they be referred to the Committee on Public Relations for a subsequent report to the Council. The motion was seconded and was carried. (See Appendix No. 4.)

There being no other business to come before the Council, it was moved to adjourn. Adjournment took place at 1:38 P.M.

ALEXANDER S. BEGG,
Acting Secretary.

APPENDIX NO. 1

REPORT OF THE COMMITTEE OF ARRANGEMENTS
APRIL 3, 1935

Mr. President and Members of the Council:

I feel that the success of the coming meeting depends in no small part upon the enthusiasm that is present or is generated in the various District Societies. As you know, the average number that has attended our Annual Meeting in recent years is approximately 1000, only about twenty per cent of our total membership.

Associated with me this year is a Committee of Arrangements that is extremely efficient and hard working, and it is our ambition that this shall be in no way just another Annual Meeting, but that it will justify its existence in every sense of the word.

To this end, letters urging attendance at the meeting have been sent to 125 new Fellows. Messages have also been sent to the Presidents of the various District Societies asking that they present to their members the benefits to be gained by each Fellow and by the local group as a whole attending our Annual Meeting.

The scientific exhibits at this year's meeting already number more than twenty-three subjects and more than thirty exhibitors. Although all the details of each exhibit are not yet available, it is apparent that many of them will be of the highest merit.

The effort this year is to have striking exhibits comprising material which is easily understood such as diagrams, models, photographs and motion pictures, in addition to exhibits of actual apparatus and technique. One of the striking features will be the exhibit on arthritis, prepared by a distinguished committee under the leadership of Dr. Robert B. Osgood, including rheumatic fever, the various types of arthritis, and the social aspects, results and methods of treatment of the various arthritic conditions. Continuous motion pictures will be shown in this exhibit which will occupy the entire space used for all scientific exhibits at the meeting two years ago.

Four members of the Lahey Clinic have developed some very interesting exhibits on thyroid disease, tumors of the lungs, and carcinoma of the colon and rectum.

Another notable feature this year will be the location of many scientific exhibits in close proximity to the commercial exhibits rather than the usual segregation of the scientific exhibits to a seldom frequented location.

On Monday morning there will be a continuous series of motion pictures, of general medical and surgical interest.

Medical Clinical Meetings have been arranged for Tuesday morning and Surgical Clinical Meetings for Wednesday morning. Both clinics are to be held at the Massachusetts General Hospital. It is hoped that the attendance will be large.

Forty-two commercial exhibit booths have been sold. The Society will receive a total of $2950.00 in payment for these booths. This is in excess of $500 more than we have ever received from the sale of commercial booths.

In fairness to the exhibitors, I would ask that you suggest to your local societies that they visit these exhibits. I think it is perhaps not sufficiently realized that the income from them is a very considerable factor in the financial support of our Annual Meeting.

We have received reports or editorials regarding their programs from the Chairmen of all the Sections. There has been this year an endeavor on the part of the Chairmen to present programs which will appeal primarily to the general practitioner and his problems.

A special effort has been made to receive and entertain the delegates from the New England States. One of our number has been assigned to receive these men. Letters of welcome have already been written to these delegates and steps are being taken to provide for their comfort while in Boston.

A feature not before attempted in Boston has been arranged this year in the form of a well-organized Ladies Committee. Headed by Mrs. William Robey, this Committee has been very active and has planned a very attractive program. Almost a year ago, Mrs. Robey arranged for a tea at the Isabella Stewart Gardner Museum.

For Tuesday evening, during our annual banquet, the Ladies Committee has arranged a supper at the Junior League and during our last day of meeting, Wednesday, a trip to Concord and a luncheon at the Concord Country Club with visits to points of interest.

WILLIAM M. SHEDDEN, *Chairman.*

APPENDIX NO. 2

The Committee on Permanent Home of the Massachusetts Medical Society respectfully submits the following report:

The note of the Boston Medical Library for $24,703.29 came due on April 1, 1935 and the treasurer of the Library requested a renewal as they were not in a position to pay off the principal of the note. After consideration between the members of your Committee and officers of the Medical Library it was agreed that a payment of $203.29 should be made on the principal, thereby reducing the note to $24,500, and the note was renewed for one year to April 1, 1936 at the same rate of interest, namely, 4½ per cent. Furthermore, it was understood that the Boston Medical Library would be in a position to reduce materially the principal of the note before April 1, 1936.

Respectfully submitted,
CHARLES G. MIXTER,
Secretary pro tem,
Committee on Permanent Home.

APPENDIX NO. 3

April 3, 1935.

Honorable Leverett Saltonstall
Speaker of the House of Representatives
State House
Boston, Massachusetts

Dear Mr. Saltonstall:

Your Committee on Rules has pending before it a bill which limits the bringing of malpractice actions against dentists and doctors to one year.

Seventy-five per cent of the malpractice suits end with favorable verdicts for the doctors. Most of the suits brought are groundless ones and a great number of them are brought after the doctors or dentists try to collect their bills.

Defendants in automobile tort cases are protected after one year. Is there any reason why doctors and dentists, who do so much good in their communities, should have claims brought against them up to the second year of the time that the services were rendered?

May we urge you as Speaker of the House to help us in the passage of this Bill?

Very truly yours,
(Signed) WILLIAM H. ROBEY.

APPENDIX NO. 4

Suggestions from Dr. Edward H. Trowbridge of Worcester and referred to Committee on Public Relations by order of the Council.

"That a committee of three—be appointed by the President of the Massachusetts Medical Society to study into the rights of the members of the Massachusetts Medical Society and their relations with the Workmen's Compensation Board.

"Considerations: 1st. Should a lay board pass on the so-called reasonable charges for services rendered by any physician or surgeon in industrial cases?

"2nd. Under what law or right does the Workmen's Compensation Board fix $50.00 as a reasonable charge for hernia operations?

"3rd. If fees for visits and operations are to be established,—let the Massachusetts Medical Society fix such reasonable charges and not a lay board.

"4th. That a physician and surgeon shall be associated with the Workmen's Compensation Board to render assistance in determining the reasonable charges.

"5th. That a committee of three—appointed by the President of the Massachusetts Medical Society—be empowered to fix such reasonable charges as will apply to all members of the Society in treating all industrial cases."

MASSACHUSETTS MEDICO-LEGAL SOCIETY

RELATIONS OF ROENTGENOLOGY TO MEDICOLEGAL PRACTICE*

BY ARIAL W. GEORGE, M.D.†

WHEN I received the invitation from your President, Dr. Gay, to discuss before your Society the relations of Roentgenology to medicolegal practice I felt, as I have so many times in the past, that because the subject was so extensive the presentation of such a paper would be very difficult as there was no beginning or ending. Those of us who have had any reasonable experience, as I have had in the past twenty-eight years, not only in the private practice of roentgenology but a very substantial part of this practice being industrial and medicolegal work, know that through these years we must have gained a certain practical knowledge and have by contact at least learned many things about the use of the x-ray. More than the importance of the x-ray, we have learned some of the difficulties one encounters in the use of the x-ray in medicolegal work. With this thought in mind and with your permission, I would like to discuss with you the many phases of this work and especially to consider some of the serious criticisms that are continually being made, perhaps a little more at the present time than in the past years particularly in the use of x-ray in industrial and medicolegal work.

The industrial and medicolegal work are identical, at least the end results are. In one instance you have a hearing before a commissioner and in the other in a higher court either before a judge or jury. I believe that what the roentgenologist needs in this type of work first of all is, if I may be permitted to use the term, mental honesty in approaching the study of the case. I am not particularly interested in the moral aspect of honesty for the moment, but I am interested in it from the practical side. Strange as it may sound, I think if a man is really going to be selfish he will be much better off to be mentally honest because this work becomes treacherous when you have a bias either for your own particular case or in your opinions expressed either written or in court against your colleagues.

I have noticed that the average roentgenologist is not well trained, nor has he had an opportunity to have that training which is obtained only through years of practical experience. In his study of a case, he puts his whole effort into finding some degree of pathology and he believes that if he is called upon to make an x-ray he must find something definitely wrong with the part under examination. You men must be aware that many x-rays are taken where no evidence of any abnormal or pathological condition is demonstrated from the x-ray regardless of

*Read at the Fall meeting of the Massachusetts Medico-Legal Society, Boston, October 3, 1934.

†George, Arial W.—Roentgenologist, Jordan Hospital, Plymouth. For record and address of author see "This Week's Issue," page 945.

the symptoms or the severity of the injury. There apparently is a strange psychology not only among roentgenologists but probably among those in other branches of medicine and that is the difficulty for one to remain unbiased where one encounters either in the passing of an opinion or in the necessity of controverting the opinion of the other and it is possible and probable that each individual builds up for his own case all the positive factors as well as the negative factors in the effort to prove himself right in court. This is unquestionably natural. It is only when positive opinions are based on unsound premises or on conditions which rarely if ever exist in the human anatomy that we encounter difficulties. It frequently happens that when a patient has sustained a fall from a considerable height and sufficient to cause an injury to a transverse process or a spinous process or to whatever part in question, that a diagnosis is passed of injury as the result of the alleged accident.

One of the most important phases of our work which causes trouble and an excessive amount of expense to the insurance companies is brought about by our written reports and the careless use of phrases, sentences or the misuse of words. This criticism I believe is just of our particular specialty and also of those in the medical profession generally. I do not believe this applies to a group of men such as those here to-day because they have been trained to be exact. However, recently I attended a hearing at which one of our medical examiners in his routine work passed certain opinions in his report of the autopsy and then on the stand was obliged to change these opinions because of the facts in the case. It was of no particular moment, but it illustrated to me as I listened to the testimony that if he had visualized his future appearance in court he would have worded his report differently and therefore would not have been obliged to change his position even to a slight extent. I have had considerable experience and I have noticed that in a routine report of a case where there was no medicolegal aspect to the case and there was no effort to build up the results of an injury, the following phrase might be used, "some hypertrophic arthritis found". In a particular instance recently, the foregoing phrase was included in a report of a spine of an elderly person who had been in an automobile accident. This term "hypertrophic arthritis found" had been used by a competent x-ray man. The orthopedic man on the case reported hypertrophic arthritis, and the lawyer in trying to build up the case attempted to point out that at least some of the symptoms could be accounted for by the excessive amount of hypertrophic arthritis and that the injury aggravated this very marked hypertrophic arthritis. I did not see the x-ray plates until I was on the stand and I did not have the opportunity of reading the actual report, but I testified that in my opinion there was not even the usual amount of hypertrophic change that should appear in an individual of this age. Therefore, my testimony differed from that of the other roentgenologist, who is a good friend of mine, and the orthopedic man, and this difference of opinion made it very unpleasant for all of us and subjected me to some criticism. All this came about because the term "hypertrophic arthritis" had been loosely used.

We experienced another difficulty as a result of our reports when we stated that "no positive evidence of fracture of the skull was found". It seemed to us that to the lay mind this was a positive statement that the x-ray was negative for fracture, but it was not so with the lawyers. I was kept on the witness stand for one hour in an effort by the attorney to make me admit that I had a mental reservation that there might be a fracture of the skull. I can see better now than I could then that there might be some question of a mental reservation. As a result of this experience, we have come to have our reports read "no fracture found by the x-ray". That leads me to say that as time goes on we use as few words as possible in our written reports. Even with this effort to have simple wording and straightforward opinions and so far as possible to the point at issue, we are met with criticism because we are in contrast with other laboratories whose reports are lengthy and yet when you sum up the reports they are pretty much the same as ours.

I doubt that you men, or even the medical profession at large, realize the tremendous increase in the number of roentgenologists in the last ten years not only in our city and state but in the country generally. Those of us who have been in the specialty for many years realize this more than the average medical man. Back in 1916 after the war started, we had recognized throughout the country only one hundred and fifty trained men with a background of reasonable experience. Immediately we were called to train one thousand men for the first army. Since then all the large hospitals in the country have special services for roentgenology and they are training their internes for this specialty in the fundamentals of x-ray, but they lack the experience which comes only as a result of years of practice. There are many small hospitals in the smaller cities and towns of our commonwealth and throughout the country generally which are first-rate hospitals and are equipped with efficient and modern apparatus but not in many instances with trained roentgenologists. These hospitals are perfectly willing either through necessity or

otherwise to take one of the general practitioners and trust and hope that he will be able to handle the situation. It does not work out well. The orthopedic man by the nature of his work assumes the responsibility of passing opinions on the x-ray work. I have always been opposed to this, principally because of the experience I had in Germany. I went over to learn something about gastrointestinal work. I found it was the clinician who was making these studies and passing the opinions. He was ninety per cent an internist and ten per cent a roentgenologist. I feel that the same is true with the great majority of our orthopedic men; they are ninety per cent clinicians and ten per cent x-ray men. I am certain that in a reasonable percentage of cases the results are not satisfactory to the patient and certainly do not prove generally satisfactory to the medicolegal practice.

One of the great stumbling blocks in the proper interpretation of x-ray films for court work seems to be the misinterpretation of congenital anomalies. It was my good fortune to spend eight years in the Department of Anatomy at the Harvard Medical School with Dr. Thomas Dwight. I have always considered this association to be one of the greatest and most wonderful experiences of my medical career. Some of you men here to-day were students of Dr. Dwight's. He, in turn, was a student of that great physician, Dr. Oliver Wendell Holmes. I do not believe that up to the time of his death there was a greater authority on anatomy than Dr. Dwight. As I said, I had the privilege of spending eight years in close contact with him in the study of congenital anomalies of the skeleton, particularly those of the hands and feet. We would take the hands and feet of cadavers and x-ray and study them. Studying the actual specimen gave us a great deal of information. I thought that, after these eight years of intensive study, I had seen every possible anomaly of the hand and foot. Yet I am frank to admit that to-day, possibly from lack of memory, we see conditions which are not familiar to us. We are, however, certain of one fact and that is that we are not applying any pathological significance to these anomalies.

The following report recently received from one of our large hospitals indicates the importance of these congenital anomalies:

"X-ray examination of your patient included the lumbosacral and sacroiliac spine. The x-ray shows a transitional left lateral lumbosacral articulation and a deficiency of the laminae of the first and second sacral segments on the left side producing an unusual type of spina bifida occulta. The spinous processes of the fifth lumbar vertebra appear to dip into the recess in the upper sacrum. The sacroiliac joints are negative."

In viewing these films, I found no evidence of a spina bifida. In fact at the time, I made the statement that it would be an unusually good film to reproduce for use in a textbook as a normal spine. In other words, the minor congenital variations reported in this case were built up to the point of calling them a spina bifida. In private practice, this would do no harm, but in a tort case or an Industrial Accident Board case or a case to be tried before the higher courts it leads to difficulty. The patient gets the report and immediately turns to a doctor and asks what it is. Naturally, the doctor familiar with this change in the vertebra says that it is a rather serious condition. From that point on it goes to the lawyer who builds up this phase. He studies the anatomy and the clinical side of the case. By the time the case reaches the court, you have a serious condition to contend with, and the entire opinion and report was erroneous and untrue.

Another term which leads to difficulty is "incomplete spina bifida". A series of cases was described by one of the men at the Mayo Clinic. This work was published and the use of the term began to appear not only in industrial work but in medicolegal practice. It was used as a definite clinical and pathological entity and in the large percentage of cases it is nothing more or less than any individual might have with or without injury.

I have tried to illustrate just briefly how careful one must be in the use of terms which have a clinical or pathological significance. I am frank to say that x-ray men are not so careful about the use of words as they should be and considerable criticism is brought about by the careless interpretation of films and the careless wording of our written reports.

We have a rather common condition found in the cervical region to which the term "spina bifida" is applied. This condition shows a wedged-shaped, incomplete formation of one or more of the bodies. In several instances I have found in medicolegal practice that a diagnosis of fracture or multiple fractures has been made. Such a diagnosis has been made by some very prominent x-ray specialists. And yet, one knows that unless disease has occurred there are no fractures possible in this region where you get a half of a body taken away without any bone reaction or callus. One can see at a glance that these changes are congenital in origin or that one may have an injury at the site of these changes which will possibly give rise to clinical manifestations.

One of the most difficult problems we have in our work, and we see it commonly in the work of others who attempt to enter the court room with their films, is the matter of distortion of the various parts under examination. False evi-

dence may be produced in the film by position or angulation and this distortion shows changes in the appearance of the vertebrae or any other part which are most difficult to understand. I have just recently been in court where I felt that the appearance of the film at issue was due to distortion and the position in which the examination was made and not the result of the alleged injury, but I had great difficulty on the witness stand where positive statements are made in bringing in any evidence of a negative nature despite the fact that the positive evidence was entirely erroneous.

Most of us have lived through the days when the term "railway spine" was so commonly used and then later the "sacroiliac" disturbance which became so popular and which was so costly in medicolegal work. I have no recollection of ever seeing a case of so-called sacroiliac dislocation which was the result of a minor injury such as stooping or lifting. On the other hand, I have seen cases where there had been severe injury and where the sacroiliac might be separated half an inch and yet the individual had none of the classic symptoms of so-called "sacroiliac" dislocation. Only yesterday I spent practically the entire day on the witness stand and part of this forenoon in an effort to prove that what one of my friends, an x-ray man, demonstrated as an abnormal condition was simply a normal process of the bones.

Another interesting fact that I have observed in our practice is that during the past few years it has become rather routine for us to be asked to go to the offices of other roentgenologists or to the x-ray departments of some of our hospitals to see the films made of a case in litigation. Strange as it may seem, I should say that eight out of every ten times that I am asked to see a set of films I differ either definitely or in some detail from the report given by the individual who examined the case. It is relatively simple in studying an x-ray to make a positive statement and have facts to substantiate your opinion whether you are right or wrong, but it is definitely difficult to disagree with a positive opinion and have facts that will prove your contention.

I have already spoken of the efforts of x-ray men to play the part of clinicians. In my opinion, if a roentgenologist goes into a case in which he is also clinician he will attempt by taking an x-ray to confirm his findings of some pathology at a given point. I have seen this done time and again and the method is rarely successful.

I believe one of the most important factors we have to consider in x-ray work is the time element relative to injury. This is of tremendous importance in determining the age of the injury and whether the alleged injury resulted from the given accident. I will illustrate this briefly: I had an opportunity of seeing a fracture of the lamina of the fourth lumbar vertebra with a dislocation of the fourth lumbar anteriorly. I asked when the injury occurred and I was told that the injury occurred in the morning and the x-rays were taken that afternoon. In studying the film, I found that the lamina showed reactionary bone changes about what would seem to be a fracture. Naturally, I felt definitely certain that this fracture was not the result of the alleged accident of that morning. The patient had sustained a fall sufficiently severe to have caused a fracture and dislocation of this vertebra, but I was quite certain and definite in my report to the insurance company that these changes were not the result of this alleged accident. Eventually, the case came to trial and the individual, a boy of twenty-three, received a rather large verdict. A year later when I was in the same court, the plaintiff's lawyer said to me that he had something very interesting to tell me regarding that trial of "so and so" last year. "After that trial," the lawyer said, "I found that this boy, when twelve years of age, was running down the street, and caught his toe in the root of a tree and fell. He was taken to the hospital and kept in a plaster cast for two years." That seemed to me rather good and definite proof that the change seen in the spine occurred at the time of that accident and not on the date of the alleged accident. One observation which I have made which may be open to question is that when you get things like fracture of the laminae and dislocations in a child under puberty you find very little bone reaction and possibly the line of fracture will be seen through life. If the same type of injury occurs in an individual of twenty-five or thirty-five, you will find more marked changes or callus formation.

In the study of x-rays in medicolegal practice, I find the time element relative to injury and also to disease which includes tumors alleged to be the result of trauma to be of the utmost importance. A simple illustration of this is a case where a man had an injury in 1922 and another injury in 1930. The case was put on service in a hospital where one of the medical directors of a large insurance company is in charge. An x-ray was made five weeks after the second injury. The physician, with all honesty of purpose, felt that the fracture was a result of the injury in 1922. When the x-ray report of 1922 was investigated, it was found to be negative relative to the vertebrae. In this instance it happened that I was on the plaintiff's side of the case. The one thing that bothered me was two definite spurs between the first and second lumbar vertebrae making

almost a complete bridging between the injured vertebrae and the one below. As I said, these x-rays were taken five weeks after the injury in 1930. I realized that too short a time had elapsed for spurs to form. Spurs started to form above the first lumbar and grew more definite over a period of a year or two so that one could study them, but the two spurs between the first and second lumbar vertebrae remained the same from 1930 to the date of trial in 1934. I was questioned for a considerable length of time about these two spurs and it rather shook my confidence a little to understand quite why they should remain stationary, but fortunately for me and to my chagrin I admit, the judge saw no reason why the man could not have had these two spurs before the injury which were not disturbed by the injury. I believe this was the answer although I did not think of it as a possibility. This illustrates again the tremendous importance of the time element in x-ray work.

There is one very troublesome feature continually arising in medicolegal practice and that is the question of arthritis. Although I do not believe many medical men to-day claim arthritis of the vertebrae to be the result of injury, yet they do make a good many extravagant claims as to the aggravation of preëxisting arthritis. In the earlier days in our Industrial Accident Board work we were inclined to report "extensive hypertrophic arthritis without evidence of injury". The plaintiff's lawyer would invariably ask if this condition was what the laymen called "rheumatism", and one would naturally say in reply "yes". And then the lawyer would question further saying, "Isn't it true, Doctor, that a man with this terrible rheumatic condition would be more disabled from now on than he would have been if he had a normal back?" And, of course, your answer would be "Yes".

As time went on, we began to realize that the word "arthritis" had no proper place in the interpretation of these changes commonly found in the backs of middle-aged people, especially those who have done laborious work. The term "arthritis" means an inflammation of a true joint. These spur formations or marginal lippings are commonly found, that is in 98 cases out of 100, around the margins of the body. Of course, as I understand it the intervertebral space is not a true joint in the sense that it has a secreting synovial membrane. The only true joint in the vertebrae is the articular process and the one part in which we rarely find disease. And so, in our Monograph on "The Vertebrae" which was published in 1928 we used the term "hypertrophic changes". Our book was abstracted in all the various languages and there was no adverse criticism given to the use of this term. It has come now to be pretty well understood that occupation and age have a definite bearing on the changes which occur along the margins of the vertebrae. Therefore, in our reports to-day we avoid using the term "arthritis".

I shall illustrate what I mean by the above. One of our medical directors and I were on a very important case. We had discussed the case and agreed not to use the word "arthritis". When on the stand, the medical director inadvertently used the term "hypertrophic arthritis". When his testimony was completed, it looked as though he had voluntarily contributed to the plaintiff's case by the use of this term because the lawyer broke the term down to mean inflammation and by adding the word "hypertrophic" he further complicated the case.

When I took the stand I decided to use the term "hypertrophic changes" in view of the other physician's testimony. When the question of aggravation arose, I said that hypertrophic changes meant no more or less than the production of normal bone and normal bone could not be aggravated or produce symptoms.

The foregoing is a very simple illustration of what is happening continually and furnishes the opportunity for false premises for changes which occur in the vertebrae.

I have several films here that are in a sense illustrative of what we are talking about. I am going to show them to you. One condition of which I wish to speak is spondylolisthesis. This condition is causing a great deal of discussion throughout the country. It is a subject of great importance in medicolegal work because it is commonly and frequently found. A careful survey of the medical literature of the world shows that it is a very occasional case of spondylolisthesis which is the result of an alleged injury. I would go so far as to say that in ninety-nine per cent of the cases it is not the result of an alleged injury, but the result of a congenital defect of the laminae in some degree of development through childhood.

Another interesting condition which causes considerable difficulty is the bipartite patella. The x-ray men as a rule do not seem to realize that you can have this condition. Invariably, they report a fracture. The only thing we can do to prove that it is not a fracture is to give the case sufficient time to see whether callus forms and sometimes you have to wait a long time. A bipartite patella will never show callus. It is, however, a condition which is easily interpreted as a fracture.

Apropos of this same condition, we find in the spine, commonly at the level of the third or fourth, usually the fourth lumbar vertebra, the remains of an epiphysis, which looks like a chip fracture. When an x-ray man or a hospital laboratory reports this condition to be a fracture

of the vertebra, perhaps in the nature of a chip fracture, it is very difficult for us to go into court and say that it is not a fracture because the x-ray evidence is characteristic of a fracture. We have tried with these cases to wait six months or a year and then x-ray them again. If the x-ray appearance of the vertebra shows no change, we know definitely that it is the remains of an epiphysis. If on reëxamination, we find callus, we know that there has been a fracture. You can see in the x-ray a dense bone on each side of the sulcus, or on each side of the fracture line. This dense bone cannot occur in three or four days' time. It takes a long period of time.

I have gone over rather hastily some of the problems and conditions that are important to us, and I don't want to take any more of your time. I hope that the discussion will open up some points that will be of interest and help to us all. I thank you.

DISCUSSION

DR. CANAVAN: Did you say that under twenty-one these fractures would not be found?

DR. GEORGE: I should say fifteen or sixteen; what I would call the average puberty, and not for the special bones, but for the average.

SPEAKER FROM THE FLOOR: I would like to ask Dr. George whether he considers a negative x-ray of the skull positive evidence of no fracture present.

DR. GEORGE: No, I do not consider any x-ray, or any series of x-rays to be negative. That is as far as we can go. The difficulty with skull x-ray work is that we, by practice, don't take all the various positions. We are trying to take the position of the base of the skull with our new curved casette. We have picked up two or three fractures of the base where we couldn't possibly do it with the ordinary series.

SPEAKER FROM THE FLOOR: Is it possible to have one line of fracture, instead of two, in the opening formed by the pubic bones into the ischium?

DR. GEORGE: I have seen what I thought was one line. Yes, I should think it could.

SPEAKER FROM THE FLOOR: Yes, but here is a solid ring. (Demonstrating manually.) Could it be one fracture?

DR. GEORGE: Yes, we pass that opinion.

DR. BRICKLEY: I would like to ask Dr. George to explain his new method of showing the fracture of the femur, as done and brought out by Dr. Leonard and Dr. Fay. Where is the x-ray film put? Where is the x-ray plate in order to show it?

DR. GEORGE: Dr. Leonard and one of the technicians worked on this; I had very little to do with that part of it. It was built to place between the groins, and the x-ray coming above the shoulder, so that you are looking down on the top of the neck of the femur. Now, in the ordinary curve that would fit in there, you would get distortion of the neck, and Mr. Parsons, who worked on the curve, tried to utilize distortion in the reverse manner; that is the only way I can say it. He made distortion bring itself back to the normal by a certain, particular curve that there is on that set. Whether it is a true anatomical view is hard to say, but it is a constant view. With the patient lying on the table, it is put in like a saddle would be, and the x-ray is brought up ten inches above the shoulder, away from the shoulder so that you are coming down through, as though I was looking down on top of my hip this way (indicating). We really can't tell anything about the fracture of the hip in the ordinary front view. I have, for years, talked about forming callus around the fracture. But when we get these views and follow them through to completion all the way through convalescence, there isn't any callus. I mean external callus; it is all internal callus, a fusion.

It has taught us that the front view on fractures of the hip joint are practically valueless, except for diagnosis; really worthless so far as position goes. You can't tell where the neck is to the head, relatively or exactly. But in this view, you have an exact standard to go by, and you can tell whether you get back to the exact anatomical position.

A STUDY OF HEART DISEASE AMONG VETERANS*

II. Analysis of a Group of Cases as to Age at Onset and Duration of Heart Disability

BY PHILIP B. MATZ, M.D.†

THE various types of heart disease make their appearance at well-defined age periods. By this is meant the causative factors produce heart disease at certain periods of the human life span. Most of the statistical data in the literature on the subject refer to the age of the cardiac patient at the time of observation rather than the time of the first appearance of heart disease.

Lamb and Turner[1] in a study of 268 patients with syphilitic heart disease found that the majority of cases occurred in the age period between the appearance of rheumatic disease on the one hand and arteriosclerosis on the other. Two-thirds of a group of 268 cases first gave evidence of cardiac symptoms between thirty-one and sixty years of age. The average age for all the cases was 45.3 years. The youngest case was twenty-five and the oldest sixty-five years of age. These observers quote the findings of six others in a study of a total of 500 cases, in which it was found that the average age at the time of the first appearance of syphilitic heart disease was 47.62 years.

White[2] in a series of 708 cases of hypertensive heart disease found that 62 per cent were in

*From the Research Subdivision, Medical and Hospital Service, Veterans' Administration.

†Matz, Philip B.—Chief, Research Subdivision, Medical and Hospital Service, Veterans' Administration, Washington, D. C. For record and address of author see "This Week's Issue," page 945.

the sixth and seventh decades; 17 per cent were over seventy years of age; sixteen per cent were in the fifth decade; 21 per cent of the cases were less than fifty years of age.

White in a consideration of rheumatic heart disease states that it usually begins between five and fifteen years of age and the height of the onset is about the tenth year.

In the discussion of arteriosclerotic heart disease, White states that the age of occurrence of the myocardial changes varies from youth to extreme old age, but it naturally increases with age. Of a group of 864 cases in New England, White and Jones found that 66.8 per cent were within the age groups between fifty and seventy years; and 26.9 per cent were in patients over seventy years of age. This type of heart disease may also occur in the young. There are cases on record in which the onset of cardiac disease occurred under thirty years of age, and, in one instance the patient was under twenty years of age.

TABLE SHOWING AVERAGE AGE AT TIME OF ONSET OF HEART DISEASE

Type of Heart Disease	Number of Cases	Age Range (Years)	Average Age (Years)
Rheumatic	93	18-56	31.8
Syphilitic	44	15-63	34.3
Bacterial infection	3	36-50	42.3
Heart disease, sequel of infectious disease	52	17-60	31.8
Thyroid	5	22-44	32.0
Cardiac neurosis	6	22-36	30.5
Arteriosclerotic	59	21-83	42.7
Hypertensive	17	21-62	35.6
General systemic disease	6	17-55	37.7
Unknown etiology	16	20-58	33.4
Potential	20	19-63	36.1
Possible	9	25-58	33.2
Total	330	15-83	34.8

In a study of the age at the time of the first appearance of heart symptoms in a group of 330 patients, it was found that the largest group 119 or 36.1 per cent gave evidence of heart disease during the age period between thirty and thirty-nine years; the next largest group 106 or 32.1 per cent showed symptoms of heart disease within the age period between twenty and twenty-nine; fifty-six or 17.0 per cent of the patients first gave evidence of heart disease within the age period between forty and forty-nine; thirty or 9.1 per cent gave evidence of heart symptoms within the age period between fifty and fifty-nine; eleven or 3.3 per cent gave evidence of heart disease prior to age twenty; and eight or 2.4 per cent of the group showed evidence of heart disease after the age of sixty.

There are certain data in this phase of the study which are worthy of emphasis. Of forty-four cases of syphilitic heart disease, thirty-two first gave evidence of the heart disability before the age of forty; in fourteen instances syphilitic heart disease appeared during the age period between twenty and twenty-nine. Among fifty-nine cases of arteriosclerotic heart disease twenty-seven or 45.8 per cent of the group first gave evidence of the heart disability before the age of forty; seven of the number had symptoms of arteriosclerotic heart disease during the age period between twenty and twenty-nine.

The average age of a group of 330 veterans at the time of onset of heart disease may be seen by referring to the table. In addition the table shows the upper and lower age limits of the patients at the time of inception of heart disease as well as the average age for each type. It will be noted that the range is from fifteen to eighty-three years for the whole group. The average age at the time of inception of heart disease was 34.8 years.

In this connection it must be pointed out that the group of heart cases under discussion is a select one. Eighty-one per cent of the patients are World War veterans of an average age of about forty-two years.

DURATION OF HEART DISEASE

Cohn[3] in commenting on the duration of heart disease refers to the study made by Romberg in a series of 176 cases, among whom he found that the interval between the date of the first heart attack, and the date of heart failure averaged 14½ months; the time between the beginning of heart failure and death averaged 5½ months, the range being from a few days to two to three years. On this basis Cohn considers the total duration as one and a half to two years with a range of from a few days to twenty years.

Cohn[4] refers to a study made by Mackie in a group of twenty-five cases of mitral stenosis in which he found that it took from one to six years for the cardiac lesion to develop. The length of time before heart failure begins is estimated by Romberg as being approximately seven years, which figure he arrived at in a study of 102 cases of valvular heart disease. The stage of heart failure lasts on an average 4.8 years.

Cohn holds that the period from the beginning of the infection to the establishment of the chronic valvular lesion is on an average four years; from this point to the onset of heart failure seven years; from here to the death of the patient an average of four years; the total period of heart disability is, therefore, in the neighborhood of fifteen years.

If one deducts four years, which is the average period from the beginning of the infection to the establishment of chronic valvular heart disease, from the total period of heart disability (fifteen years), then one arrives at the

figure of eleven years, which is the average duration of heart disease from the date of diagnosis to the date of death.

Grant[5] in a study of 1000 cardiac patients found that 51 per cent were known to be alive at the end of ten years, and 47 per cent were known to have died within this period. This observer found that the average life in cases of syphilitic heart disease with aneurysm is over five years; and in cases of syphilitic heart disease without aneurysm 58 per cent died within ten years. The general view among American observers, however, is that the average duration of life from the date of diagnosis to date of death in syphilitic heart disease is two to three years.

The writer[6] in a study of the duration of cardiovascular disease in a group of 491 cases found that the cardiac disability was present for periods varying from one to six months in thirty cases to from eight to nine years in forty-one cases; the largest group, eighty-five in number, gave a history of having had heart disease from seven to eight years. In the same study the writer estimated the duration of various cardiovascular lesions in 252 patients who died from heart disease. The interval between the date of diagnosis of the heart lesion to the date of death of the patient was as follows: For acute dilatation of the heart, eight months; acute endocarditis, four months; chronic endocarditis, four years; chronic myocarditis, four years; combined aortic and mitral disease, five years; and mitral insufficiency, three years.

For the purpose of arriving at an estimate of the survival period in the living cardiac cases and the duration of heart disease in the deceased cases, the data of 330 of the 500 veterans were analyzed. Of the 330 patients, 286 were living and forty-four were deceased.

SURVIVAL PERIOD OF ETIOLOGICAL TYPES OF HEART DISEASE

In a study of the survival period of 286 living patients with cardiac disease, it was found that the largest group consisting of 105 patients or 36.7 per cent of the total number, gave a history of having had heart disease from ten to fourteen years; the next largest group, eighty-seven, or 30.4 per cent, had heart disease from one to four years; twenty-two or 7.7 per cent of the group gave a history of having had heart disease for periods less than one year; and twenty-seven or 9.5 per cent gave a history of having had heart disease for fifteen years or longer.

In a study of the survival period of the living veterans affected with various etiological types of heart disease it was found that of eighty-eight patients with rheumatic heart disease, the largest number, thirty-two, had a survival period of ten to fourteen years; and the next largest number, twenty-one, had a survival period of one to four years.

Of thirty-one cases of syphilitic heart disease, the largest number, thirteen, had a survival period of one to four years; and the next largest number, seven, had a survival period of five to nine years.

Of fifty cases of heart disease, the sequel of infectious diseases, the largest number, twenty-eight, had a survival period of ten to fourteen years; and the next largest number, twelve, had a survival period of one to four years.

Of forty-six cases of arteriosclerotic heart disease, the largest number, twenty-one, had a survival period of one to four years; and the next largest number, eleven, had a survival period of ten to fourteen years.

Of fifteen cases of hypertensive heart disease, the largest number, six, had a survival period of ten to fourteen years; and the next largest number, four, had a survival period of one to four years.

In connection with the above data, it is desired to point out that the survival period of the living cardiac patients is from the date of the inception of heart disease to the date of this study. Accordingly, the data represent the minimum figures.

SURVIVAL PERIOD OF ANATOMIC TYPES OF HEART DISEASE

A study was made of a group of cases to show the survival period of heart disease by anatomic types. For this purpose it was necessary to select patients with a single cardiac lesion. It was not possible to find any deceased cardiac patients of this type. However, seventy-one living patients gave evidence of a single anatomic heart lesion.

Of the seventy-one living cardiac patients with a single anatomic lesion thirty-three have survived periods of from ten to fourteen years; and twenty-one from one to four years.

Of twelve cases with hypertrophy of the heart, four have survived from one to four years; and six from ten to fourteen years. Of twenty-one patients with chronic myocarditis the largest number, ten, have survived periods from ten to fourteen years. Of twelve cases of mitral insufficiency, eight have survived periods from ten to fourteen years. Of ten cases of mitral stenosis, four have survived periods from one to four years; three from five to nine years; and three from ten to fourteen years.

DURATION OF HEART DISEASE

A study was made of the duration of heart disease among forty-four deceased veterans. It is realized, that the group is a small one, nevertheless, it throws light upon certain important phases of the subject. The largest group, consisting of seventeen patients or 38.6 per cent of the total number, showed a duration of heart disease from one to four years; and the next largest number, consisting of ten patients or

22.7 per cent of the total number, gave a history of heart disease of ten to fourteen years' duration; two of the forty-four patients had heart disease for a period of fifteen years or over. The average duration of the heart disability for the total group of forty-four deceased veterans was 5.7 years.

A study of the duration of the etiological types of heart disease indicates that the average duration of five cases of rheumatic heart disease was 9.8 years; thirteen cases of syphilitic heart disease had an average history of heart disease for five years; in thirteen cases of arteriosclerotic heart disease the average duration of the disability was 6.25 years.

SUMMARY AND CONCLUSIONS

1. A group of 330 cardiac patients were carefully studied for the purpose of ascertaining the age at the time of onset of heart disease. It was found that the largest number consisting of 119 patients or 36.1 per cent gave evidence of the inception of heart disease during the age period thirty to thirty-nine years; the next largest group, 106 in number or 32.1 per cent, showed symptoms of heart disease within the age group twenty to twenty-nine. Certain data regarding the age at the time of inception of heart disease are worthy of emphasis. Thirty-two of a group of forty-four cases of syphilitic heart disease gave evidence of a heart disability before the age of forty; in fourteen instances syphilitic heart disease appeared within the age group twenty to twenty-nine. Among fifty-nine cases of arteriosclerotic heart disease twenty-seven or 45.8 per cent first gave evidence of disease of the heart before the age of forty; in seven instances of arteriosclerotic heart disease symptoms were noted within the age period twenty to twenty-nine. The average age at the time of inception of heart disease for the whole group was 34.8 years. The above data must be considered in the light of the fact that this group of patients is a select one and that 81 per cent of the number were World War veterans of an average age of forty-two years.

2. In a study of the survival period of 286 living patients with heart disease it was found that the largest number, 105 or 36.7 per cent, gave evidence of the existence of heart disease from ten to fourteen years. Twenty-seven of the number survived the heart disability for fifteen years or over.

3. In a study of the survival period of cardiac patients with anatomic types of heart disease a selection was made of seventy-one living patients with a single cardiac lesion. Cases of cardiac hypertrophy, chronic myocarditis, mitral insufficiency, and mitral stenosis, showed varying survival periods up to fourteen years.

4. Among forty-four deceased veterans with cardiac disease the average duration of the heart disability was 5.7 years. The average duration of rheumatic heart disease in the latter group was 9.8 years; syphilitic heart disease had an average duration of five years; and arteriosclerotic heart disease had an average duration of 6.25 years.

5. The survival period of the group of living cases appears to be longer than the experience in civil life. The reason for the same is no doubt in part due to the availability of Veterans' Administration hospital facilities for the treatment of cardiac patients, and the ease with which such hospitalization is obtained.

REFERENCES

1. Lamb, Albert R., and Turner, Kenneth B.: Cardiovascular syphilis. Nelson Loose-Leaf Living Medicine. 4: 337, 1924.
2. White, Paul Dudley: Heart Disease. New York: The Macmillan Company. 1931.
3. Cohn, Alfred E.: Heart disease from the point of view of the public health. Am. Heart J. 2: 386, 1926-27.
4. Cohn, Alfred E.: Heart disease from the point of view of the public health. Am. Heart J. 2: 275, 1926-27.
5. Grant, R. T.: After histories for ten years of a thousand men suffering from heart disease. (A study in prognosis.) Heart 6: 275 (June) 1933.
6. Matz, Philip B.: Statistical studies bearing on problems in the classification of heart diseases. V. Heart disease among ex-service men. Am. Heart J. 4: 313 (Feb. and April) 1929.

MEDICAL SOCIAL SERVICE DEPARTMENTS

Five of the medical social service departments of the Boston hospitals continued to report on the uniform statistical forms for March. Figures showing the number of patients receiving social treatment and minor services for the last three months are given in the table below. The social service departments of the City Hospital and the Massachusetts General Hospital reported a larger number of cases, both intensive and incidental service cases, in March than in either of the two preceding months. In connection with relief expenditures of voluntary agencies it should be noted that the social service departments furnish medical relief such as special appliances, special diets and convalescent care. During February four of the departments spent $1805 for 585 patients for medical relief.

NUMBER OF CASES UNDER CARE OF FIVE BOSTON MEDICAL SOCIAL SERVICE DEPARTMENTS, JANUARY TO MARCH, 1935

Departments	Under Care			Minor Services		
	Mar.	Feb.	Jan.	Mar.	Feb.	Jan.
Beth Israel	117	124	129	294	284	318
Boston Dispensary	361	360	335	708	561	611
Children's Hospital	108	107	114	204	268	231
Boston City Hospital	765	736	720	5667	4833	4579
Massachusetts General Hospital	472	444	435	1346	1089	1154

—Review of Social Statistics. Boston Council of Social Agencies.

MEDICAL PROGRESS

RECENT PROGRESS IN PHYSIOLOGY

BY PERCY G. STILES, PH.D.*

THE Nervous Government of Breathing.—Sir Joseph Barcroft's "Features in the Architecture of Physiological Function" is one of the most fascinating of scientific books[1]. Many of the author's clear and stimulating expositions bring home to the older reader a realizing sense of the swiftly changing conceptions of bodily processes which are the result of late researches. This is conspicuously true in the physiology of respiration. The traditional teaching has long been to the effect that there is a centre concerned with the regulation of breathing and that this centre is definitely localized and sharply circumscribed. Its position in the medulla has been confidently pointed out; it has been spoken of as single or, at most, as consisting of right and left halves with reference to the mid-line. This is the "vital knot"—noeud vital—of classic physiology.

In the light of many studies reviewed Barcroft concludes that the "centre" of the textbooks does indeed exist but that it is a rather primitive and inflexible mechanism. If it is deprived of the modifying influence of certain other centres normally coöperating with it, the breathing observed consists of a series of convulsive gasps. The intervals between these movements are long and irregular. Each act is a swift and profound inspiration which seems to involve a maximal release of the available muscular power. The collapse of the chest which instantly follows is said to be wholly passive in nature; no use is made of muscles having a potential expiratory effect. The noeud vital, viewed in reference to this performance, is the "gasping centre."

As has been said, the activity of the gasping centre is ordinarily modified by influences from higher levels. According to the evidence there is a source of such directive impulses as far up as the anterior part of the pons. This has lately become known as the pneumotaxic centre. It affects the gasping centre in at least two ways: it cuts off the discharge to the inspiratory muscles before they have been maximally contracted and it introduces an active muscular component into the expiratory movement. It also quickens the rhythm of the breathing above the slow rate characteristic of the gasping centre when left to itself. The pneumotaxic centre is probably spurred to recurrent action by sensory currents. Included among them are those long recognized as originating in the lungs as they are alternately stretched and relaxed. These ascend by way of the vagi. Others of related function may come from the muscles used in breathing.

Under certain conditions, pathological or experimental, there may be suggestions of a mechanism at work which seems to be intermediate between the pneumotaxic and the gasping centres. The tendency disclosed is to hold the breath at the summit of each inspiration. A type of breathing results in which there are prolonged inspiratory tetani. This is described as apneusis. It is oddly suggestive of a procedure calculated to make the lungs capacious reservoirs to hold reserve air for emergencies, in diving, for example.

Concerning the Whale.—Turning to another section in Barcroft's book we find some arresting considerations respecting the whale[2]. The mass involved in the physiology of this animal is something wholly unfamiliar. The energetics and metabolic tables must be upon the same scale. We are told that a whale with a length of 80 feet may weigh 120 tons. Within such a body, comparable to the hull of a tow-boat, there may circulate some 8000 liters of blood. The heart which drives it has the weight of a horse, say 1300 pounds. The oxygen capacity of the blood is estimated to be 1700 liters. This is enough to meet the resting requirements of 1700 men for about four minutes.

Now it happens that such a whale has actually the mass of about 1700 men. The animal can remain submerged for something like half an hour. Its superiority to the human diver is therefore marked and the most obvious explanation is found in an economical metabolism. The basal rate for the whale, referred to weight or cubic contents, may be no more than one-tenth of the rate for man. It may be objected that the whale maintains a high body temperature though living in cold water. This possibility is perhaps sufficiently accounted for by two facts. First, the surface of the huge animal is relatively small, and, secondly, the superficial tissues (blubber) afford an efficient barrier to rapid heat loss. It is stated that the carcass of a whale scarcely cools after death, the heat of decomposition soon being substituted for that of normal metabolism.

Other problems raised by the proportions of the whale have to do with the power necessary to its progress through the water. They are much like those dealt with by the designer of submarines. Such an expert has coöperated with Barcroft in this connection; according to his figures a whale developing 7.5 horse power can swim below the surface at a rate of 5 knots an hour. Breathing must be performed as often as once in five minutes, or at the end of every mile. To attain to a rate of 10 knots the horse power must be stepped up to 53 and for 15 knots

*Stiles, Percy G.—Assistant Professor of Physiology, Harvard University. For record and address of author see "This Week's Issue," page 945.

168 horse power will be required. Acceleration is therefore costly and so also is swimming in broken water at the surface of the sea.

Synthetic Powers of Animal Cells.—In the teaching of elementary biology it used to be customary to assign all synthetic activities to the higher plants and to regard animal cells as wholly destructive. This sweeping distinction has long been recognized to be untenable. There are innumerable instances of the combination of simpler into more complex substances by animal protoplasm. It remains of interest to find out the extreme limits of such synthetic capacities. A contribution bearing on this question has been made by Mast and Page[3]. The attempt has been to determine the most meagre assortment of raw materials from which a protozoan—Chilomonas—can build up its own substance with resulting growth of the culture.

The organism multiplies in a medium containing $MgSO_4$, K_2HPO_4, sodium acetate and glycine. Ammonium chloride may be substituted for the glycine. If we are not permitted to claim that radiant energy is utilized this would seem to be hard fare.. Even bacteria which we regard as plants have need of compounds of a similar order.

Alcohol and Muscular Work.—According to the present conception the oxidation of organic fuel is essentially subsequent to muscular activity and restorative in function. It is held that the three chief types of food are all available for this purpose. Whether alcohol may take its place with them is evidently a matter of great interest. It has been investigated by Canzanelli, Guild and Rapport[4]. Their inferences as to the rate of oxidation of alcohol in the experimental animal (dog) are based on measurements of the respiratory quotient. This ratio has a lower numerical value for alcohol than for any of the other foodstuffs. The indication is clear that the resting animal burns alcohol rather rapidly after ingesting it.

Comparing the condition of rest with that of exercise after alcohol the authors fail to find that this compound can serve as a source of muscular energy. Alcohol, from this point of view, figures as a "respiratory" food in the sense in which Liebig long ago used the term. Its energy is not wasted, it is at least available for the maintenance of the body temperature. When it is used for such a purpose it spares other types of food from consumption and thus may conserve them for service as muscle fuel.

Voluntary Increase of Metabolism.—This subject is discussed by Carpenter, Hoskins and Hitchcock[5]. Measurements of oxygen consumption and related observations were made on one of the workers during many short periods. usually of ten minutes each. The majority of these periods were designed to fulfil the conditions aimed at in determining basal metabolism; bodily relaxation and mental indifference were so far as possible realized. From time to time the subject was directed to concentrate upon securing increased intensity in the metabolic process. According to his analysis of the mental procedure involved in this there was no conjuring up of an emotional situation. Muscle tonus was annulled excepting for one instance in which it was permitted to develop. External signs of activity were of the slightest.

When the voluntary effort to speed up metabolism was made the objective results were considerable. The increase of oygxen consumption was from 13 to 32 per cent. Pulse rate accelerated by 17 to 26 per cent and there were increments of a similar order in blood pressure, both systolic and diastolic. These are just such changes as would attend a mild degree of excitement. They have been induced, however, in connection with a state of mind hardly to be described in these words. It would rather be spoken of as a fixation of attention or exercise of the will. When we consider that falling asleep vitiates a metabolism test by lowering the values obtained it may be said that we have here the effects radiated to the tissues from a brain that is acutely awake. The most emphatic sign that a period in a test should be viewed with suspicion or thrown out is doubtless a quickening of the pulse.

Diabetes Insipidus.—This remarkable disturbance of the water balance has long been referred to lesions of the pituitary or of parts of the brain in its neighborhood. It has been difficult to appraise the several factors which may be concerned. A paper by Richter deserves to be cited as contributing to the solution of the problem[6]. Operations were performed upon the pituitary in rats, effects on water economy were noted, and autopsies were utilized to determine the residual state of the gland. In general, it appeared that two conditions must coexist to maintain a typical diabetes insipidus. The posterior lobe must be destroyed; the anterior lobe must not be too badly damaged.

The inferences of Richter from the experimental facts are guarded and tentative. It is suggested that the primary source of the disturbance is the absence of the normal antidiuretic substance liberated by the posterior lobe. With this check withdrawn the kidneys secrete a great volume of water. Thirst is secondary: it is the reaction by which the animal compensates for this loss. The rôle of the anterior lobe is more in doubt. It might be held to oppose the posterior lobe as respects the secretion of urine. A less specific action may be conceived; when the organism is deprived of the anterior lobe there is such a widespread depression of its capacities that the water exchanges would not be expected to continue on a high level.

The importance of the stalk should be mentioned. Richter points out that it may contain the channel through which the antidiuretic product of the posterior lobe takes its departure.

It is a fact that cutting the stalk may cause polyuria. Under these circumstances the hormone may fail to be distributed. An alternative conception may represent the stalk as including nerve paths by way of which the lobe is aroused to action. Severing the stalk would, in this case, leave the posterior lobe in some degree paralyzed. This view would also open the question whether there are definite centres in the adjacent brain-stem which can be made responsible for stimulation of the lobe.

Breathing Pure Oxygen.—It is usually stated that the substitution of oxygen for air as breathed by a resting subject has no clear effect on the metabolism or on the feelings of the individual. Inhalation of oxygen at atmospheric pressure may slightly improve some types of athletic performance. It decidedly prolongs the possible duration of holding the breath. It was long ago observed that animals made to breathe pure oxygen under excessive pressure might be injured in consequence. Sometimes they collapsed abruptly; in other cases pneumonia developed after the trial.

The experiments now to be reviewed are those of Behnke, Johnson, Poppen and Motley[7]. Human volunteers were studied. Continuous breathing of oxygen at one atmosphere could almost always be carried on for four hours without disturbing effect. It may be noted that the pressure of oxygen in this case is nearly equal to that in ordinary air compressed five-fold, such air as must be furnished to a diver about 170 feet below the surface. Of course the breathing of oxygen at standard pressure presented no technical difficulties. To breathe it at the higher pressures employed (two to four atmospheres) recourse was had to the large pressure chamber belonging to the Harvard School of Public Health.

Pressures of two and three atmospheres were well borne, the former for three and the latter for two hours. Oxygen at four atmospheres appeared definitely hurtful. Only two men attempted to breathe it and both were prostrated after about forty-five minutes. Loss of consciousness occurred without any premonition; in one subject the experience seemed to be that of simple fainting, in the other there was a convulsive seizure and twenty minutes passed before complete recovery. Symptoms of pulmonary irritation were reported in only one case and, oddly, when the gas was furnished under atmospheric pressure. The effects were not at all severe.

Under pressures of the order of four atmospheres so much oxygen dissolves in the blood that the supply to the tissues would seem to be adequate even apart from the service of hemoglobin. Earlier writers have pointed out that when the corpuscular mass is out of action the elimination of carbon dioxide may be hindered. Under ordinary conditions there is a transformation of reduced hemoglobin to oxyhemoglobin in the lungs. This is equivalent to the addition of acid to the blood and it tends to expel carbon dioxide. If, however, there is no reduced hemoglobin to be oxidized in the lungs because the corpuscles have not been desaturated, this expulsive action will be missing. The carbon dioxide of the arterial blood should, theoretically, increase and toxic effects resulting from high oxygen pressure may possibly be explained in this connection.

The Colon.—Raiford and Mulinos have conducted an interesting research on the "exteriorized" colon of the dog[8]. A section of the transverse part is brought out through an incision and grafted upon an area from which the skin has been removed. The vascular and nervous connections are preserved. The loop is slit lengthwise and opened so that it lies flat on its new base with its mucosa exposed. The direction now maintained by the longitudinal and circular muscle coats is clear. Records of their contractions are made. Attention has been directed chiefly to analyzing the sequence of events following mechanical stimulation of the intestinal lining.

The movements observed are classed as local reflexes. Though the central connections of the region were originally intact, as stated above, the motor reactions were not affected by their later interruption. It was found, in brief, that friction upon the mucosa caused contraction of longitudinal elements below the point stimulated and, after the lapse of three to five seconds, contraction of circular bundles above. The effect, in the intact colon, would be to form a pouch below the stimulated zone and to press into it material from higher up. This corresponds with the myenteric reflex of Bayliss and Starling so far as the visible result is concerned. The explanation offered is simpler than the traditional one inasmuch as no inhibitory phases are postulated. The mediation of the stimulus is held to be accomplished in two stages. The first consists in a radiation of nerve-impulses to localities in which a humoral agent is set free; the second comprises the diffusion of this hormone to the contractile cells.

REFERENCES

1. Barcroft, J.: Features in the Architecture of Physiological Function. New York: The Macmillan Company, 1934. Chapter I.
2. Ibid: Chapter V.
3. Mast, S. O., and Page, I. H.: Protoplasma. 20: 326, 1933.
4. Canzanelli, A.; Guild, R., and Rapport, D.: The use of ethyl alcohol as a fuel in muscular exercise. Am. J. Physiol. 110: 416, 1934.
5. Carpenter, T. M.; Hoskins, R. G., and Hitchcock, F. A.: Voluntarily induced increases in the rates of certain "involuntary" physiological processes of a human subject. Am. J. Physiol. 110: 320, 1934.
6. Richter, C. P.: Experimental diabetes insipidus: Its relation to the anterior and posterior lobes of the hypophysis. Am. J. Physiol. 110: 439, 1934.
7. Behnke, A. R.; Johnson, F. S.; Poppen, J. R., and Motley, E. P.: The effect of oxygen on man at pressures from 1 to 4 atmospheres. Am. J. Physiol. 110: 565, 1935.
8. Raiford, T., and Mulinos, M. G.: The myenteric reflex as exhibited by the exteriorized colon of the dog. Am. J. Physiol. 110: 129, 1934.

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

EDITED BY RICHARD C. CABOT, M.D.

CASE 21201

PRESENTATION OF CASE

A thirty-two year old single Greek cook was brought to the Emergency Ward in a stuporous condition. (The history is somewhat unreliable.)

Approximately eight months before entry the patient suffered a head injury during an automobile accident. He was not unconscious but remained in bed for three weeks and then went to work for two weeks. Since the accident he had almost continual headaches and also episodes that he described as convulsions. Three weeks before entry he had a profuse nosebleed lasting several hours. This, in addition to his headaches, caused him to go to a hospital where he remained for ten days without improvement. He was unable to remember anything between the time of his discharge from the hospital, ten days ago, and his entry here. On the day before admission he was found in a stuporous condition by some friends. He had a convulsion and a nosebleed.

A note from the hospital where he had been for ten days previous to entry stated that he had epileptiform seizures every half hour and that his friends had said that he had been having convulsions for two days prior to that entry. Laboratory findings during his stay in the hospital showed a nonprotein nitrogen of 200 milligrams, and urine that contained a large trace of albumin and 30 to 40 white blood cells in an uncentrifuged specimen. A lumbar puncture was reported as being normal. The blood pressure was 144/94. A cystoscopy revealed cystitis for which he received hot boric bladder washes. He was placed on constant drainage but because of his inability to coöperate was discharged against advice.

Sixteen years before entry an abdominal operation was performed in England. Before this operation he had complained of vomiting and upper abdominal pain. Thirteen years before entry the patient fell through a hatchway and landed on his left side injuring, he believed, his left kidney. He remained in bed for about thirty days and then entered a hospital where his left kidney was removed. He had gonorrhea sixteen years before entry. During the past two years he had nocturia three to five times.

Physical examination showed a stuporous, well-developed but undernourished man in no acute distress. The eye grounds were normal. There was a bleeding lesion at the angle of the mouth and several granulating oozing lesions over the lower extremities and one in the left groin. His mucous membranes were pale. His teeth were dirty and carious, with marked pyorrhea. The inguinal glands were moderately enlarged. His heart was enlarged, the left border of dullness being nine centimeters from the midsternal line, 1.5 centimeters outside the midclavicular line. There was a soft systolic murmur at the apex. The blood pressure was 140/90. There was a long upper left rectus and a left flank scar.

The temperature was 98.2°, the pulse 72. The respirations were 18.

Examination of the urine showed a specific gravity of 1.006 to 1.014 with a large trace of albumin. The sediment contained 15 to 20 white blood cells, an occasional red blood cell and granular cast. A small amount of Bence-Jones protein was found in two urine examinations. The red blood cell count was 3,100,000, with a hemoglobin of 50 per cent, the white cell count 6,200, 66 per cent polymorphonuclears. One stool examination gave a questionable positive guaiac. The nonprotein nitrogen of the blood was 175 milligrams. The plasma protein was 5.5 milligrams, the chlorides 107 cubic centimeters of N/10 Cl. Creatinin was 6.6 milligrams per cent. The serum calcium was 6.35 milligrams per cent and the serum phosphorus 5.70. A Hinton test was negative. A lumbar puncture was negative.

X-ray examination of the skull showed a one centimeter area of decreased density located just above the left frontal sinus. A flat abdominal plate showed the absence of the left kidney and no evidence of disease of the bones. There was a congenital variation of the left lateral transverse process of the first lumbar vertebra.

The patient acted confused. He was given two liters of 5 per cent glucose in saline every day. His urine output increased slightly. He became more rational and less drowsy. His nonprotein nitrogen fell to 127. A cystoscopic examination showed enlargement of the right kidney but no deformity of the pelvis. Three weeks after admission he developed pain in the right ear and a few days later the drum ruptured spontaneously, following which the pain disappeared. During the sixth week he became irrational, had six convulsions and died.

DIFFERENTIAL DIAGNOSIS

DR. MYLES P. BAKER: By the time of his first hospital admission elsewhere such possibilities as subdural hematoma or rapidly progressing brain tumor, with symptoms of increased intracranial pressure, or some more intangible result of the accident—all these possibilities can

be excluded. The picture of uremia was definitely manifest. However, this is an atypical picture of renal failure. At once the possibilities that may be considered with atypical cases of chronic nephritis come to mind.

There are certain points in this man's clinical picture that are typical of uremia from any cause. The decrease in capacity of the kidneys to concentrate the urine, the elevation of the nonprotein nitrogen and the other findings in the blood chemistry,—the retention of creatinin, the indication of inability to excrete chloride and phosphate; the decrease in serum calcium,—all of these are consistent with the diagnosis of uremia. The spinal fluid pressure is typically not increased in these cases of uremia without papilledema. We might presume that the CO_2 combining power of the blood was decreased. I cannot vouch for the skin lesions except to suggest that they may have been infected excoriations on the very itchy skin of a uremic patient. Typically patients with uremia have bleeding lesions around the gums and rather intractable nosebleeds. On the other hand, the absence of edema, the absence of unequivocal evidence of hypertensive retinopathy, of an important degree of hematuria, and of definite evidence of cardiac hypertrophy in the face of hypertension are rather unusual absences if we deal with that most common cause of uremia, chronic glomerulonephritis.

But we have in this man the unusual finding of a small amount of Bence-Jones protein in two specimens. This finding I believe was established by Dr. Jacobson and he tells me that it was described as a minimal amount of Bence-Jones protein. Is Bence-Jones proteinuria ever found in chronic glomerulonephritis? As far as I can make out, no. An occasional report may have drifted into the papers on the subject but then its reliability has been questioned. Can we exclude the commonest cause of uremia simply because he has no hypertension, because he has no marked hematuria, because he has no hypertensive retinitis? We cannot. Occasionally I know there have been cases reported at autopsy showing typical chronic glomerulonephritis without the usual hypertension and retinopathy. The fact that this man by pyelogram showed an enlarged right kidney is rather against the diagnosis of the end stage of chronic glomerulonephritis, however. Similarly, the absence of hypertensive retinitis or definite evidence of marked cardiac hypertrophy and dilatation would pretty definitely rule out vascular nephritis in this case, even though we had no Bence-Jones protein present at all.

Does his past history help us at all? We know he had, years ago, a left nephrectomy a month after an accident. It might have been some pyemic lesion in the kidney, possibly a "pus kidney", a renal stone, or a large polycystic kidney. The latter sometimes used to be removed. The pyelogram, which was taken because of the obscure etiology of this man's uremia, does not show the elongation and deformity of the major and minor calices that we see in polycystic kidney.

That leaves us with three situations that might contribute to this man's renal failure: (1) chronic pyelonephritis, without obstruction in the genito-urinary tract, such as has been described by Dr. Longcope, (2) the destructive changes in the kidney noted as a complication of long standing hyperparathyroidism, and (3) the "Bence-Jones kidney" which is shown in those cases of multiple myeloma which develop renal failure. Several, four of nine, of Dr. Longcope's cases of chronic pyelonephritis have a similar absence of hypertension, retinal vessel changes and cardiac hypertrophy; and this man's urinary sediment is in keeping with such a condition, with few casts, notably. But almost all of the cases had small kidneys with more evidence of infection of the kidney pelvis than I should say was present in this man's film.

Is it possible that this man might have had long standing hyperparathyroidism going on ultimately to what Dr. Albright has called a nephrocalcinosis and renal failure, and seen terminally when the blood calcium and phosphorus studies could only be non-specific, such as one finds in rapidly developing insufficiency of functioning renal units? It is unlikely, and the question at once comes to mind whether you can have Bence-Jones proteinuria in hyperparathyroidism; recently Dr. Albright has stated that such is the case, that the protein was found in small amounts in two cases of hyperparathyroidism in which there had been marked bone changes. In view of the absence of evidence of von Recklinghausen's disease in this man, the duration of whose disease must have been long, it seems to me very unlikely that we are dealing with hyperparathyroidism. Such hyperplasia of the parathyroid glands as there may be is possibly secondary to a chronic pyelonephritis.

That leaves us with the possible diagnosis of chronic pyelonephritis in association with more diffuse myelomatosis than one would gather from the x-ray evidence. We know that the "Bence-Jones kidney," so-called, is not necessarily a small kidney; we are dealing with a slowly developing atrophy of kidney tubules as a result possibly of a marked dilatation of the tubules by plugs of inspissated protein material thought to be Bence-Jones protein. An unusual type of hydronephrosis results. On the basis of averages, 90 to 95 per cent of the cases that show Bence-Jones protein in the urine will have multiple myeloma, 3 to 5 per cent will have leukemia involving the bone marrow. That seems to be excluded in this case; so that our choice lies between multiple myeloma with the chronic intrarenal hydronephrosis seen in that situa-

tion, or a much less likely etiology. If we are to make one diagnosis I shall suggest that this man has myelomatosis and "Bence-Jones" kidney.

Clinical Discussion

Dr. Bernard M. Jacobson: In view of the very high incidence in this hospital during the past two years of cases showing Bence-Jones protein in the urine and the high correlation with the histologic demonstration of myeloma, I knew that we sooner or later would meet our Waterloo if we stuck too strongly to the thesis that Bence-Jones protein always indicates myeloma. If it had not been for the curiosity of Dr. Kranes I might still rest on our previous records of Bence-Jones proteinuria. Dr. Kranes picked it up in this patient and I confirmed this finding. I merely want to state that all the cases of myeloma have excreted the protein in a three plus degree. This patient's was one plus, and it is quantitatively similar to the amount found in one of Dr. Albright's cases of hyperparathyroidism. The cases that have been reported in the literature of non-myeloma causing Bence-Jones excretion have also been one plus degree.

Dr. Arlie V. Bock: I should be inclined to make a diagnosis of chronic glomerulonephritis and throw out the Bence-Jones.

Dr. Walter Bauer: I think I should make a diagnosis of chronic glomerulonephritis in view of the fact that the Bence-Jones proteinuria has always been reported as being only very slight. I do not think there is sufficient evidence to entertain the possibility that we are dealing in this instance with a kidney lesion (nephrocalcinosis) secondary to hyperparathyroidism. Even though this patient represents uremia secondary to the nephrocalcinosis of hyperparathyroidism, we would still expect to find a hypercalcemia, with a hyperphosphatemia, instead of the usual hypophosphatemia. In other words, in such instances there would be an elevation of the inorganic serum phosphorus and in consequence a hyperphosphatemia. I believe that there may very well be an unrecognized complication. It would help considerably if we knew the exact reason for the previous nephrectomy. He may have been suffering from a hematuria caused by an acute glomerulonephritis which was interpreted as being due to traumatic rupture of the left kidney and in consequence nephrectomy was advised.

Clinical Diagnosis

Uremia.

Dr. Myles P. Baker's Diagnosis

Myelomatosis and "Bence-Jones" kidney.

Anatomic Diagnoses

Chronic glomerulonephritis.
Operative wound: old nephrectomy, left.
Cardiac hypertrophy.
Dermatitis, acute and chronic.

Pathologic Discussion

Dr. Tracy B. Mallory: The history in this case is certainly strewn with red herrings in the form of Bence-Jones protein and the skull defect with its sharp margins. The skull defect turned out to be a complete absence of the inner table over a quite localized area, simply an outer table of the skull at that spot and nothing else, no tumor. Sections of bone marrow from several bones show no evidence of myeloma, although that diagnosis was seriously entertained in the House as well as by Dr. Baker.

The remaining kidney was not enlarged and I think Dr. Baker was misled by that statement. The pelvis, it is true, was enlarged but the kidney substance itself was small; it weighed 125 grams which would be a little small for a single kidney in a normal adult man and is extremely small for a single kidney where the other kidney had been removed four or five years before. The remaining kidney should have hypertrophied to at least 200 grams in the intervening time. The capsules stripped with a great deal of difficulty and left a finely granular scarred surface and microscopically the lesion is typical of a chronic glomerulonephritis.

The heart, in spite of the clinical evidence to the contrary, was considerably hypertrophied, weighing 450 grams. So that there are enough misleading data to make the diagnosis extremely difficult.

There are two points in relation to the differential diagnosis which have not been mentioned but which seem to me to be of some value. Our cases of true myeloma with "myeloma kidneys" have all shown high serum protein figures, whereas this was low. Then as regards the atrophic kidney of long standing hyperparathyroidism, such kidneys always show on histologic examination extensive calcium deposits in the parenchyma and even in the form of casts in the tubules. In the two well marked examples we have seen, these deposits have shown up very clearly in x-ray plates of the kidney region, whereas in this case the kidney outline could not even be visualized.

CASE 21202

Presentation of Case

A thirty-six year old American housewife entered because of uterine bleeding.

Approximately two and a half months before entry the patient believed that she was preg-

nant because she went one day over her expected menstrual period and attempted to induce an abortion with a boiled curved instrument. She repeated this process for several days and following each attempt there was slight bleeding. At no time was there anything resembling blood clot or tissue. The blood staining disappeared after several days but recurred once the following week for about half an hour. There was no further bleeding until six weeks before entry, when she bled for nine days. The bleeding was quite profuse but disappeared following bed rest. Two weeks before entry the bleeding started again and lasted until admission. For the first week it was slight but gradually became more profuse and contained clots. At no time during the illness was there fever, malaise or pain.

The family history is non-contributory.

She had been married fifteen years. Her husband and four children were living and well. Her last pregnancy was eight years before entry. There were no miscarriages.

Her past history, including her menstrual history, is negative.

Physical examination showed a well-developed and nourished, slightly pale woman. The heart and lungs were negative. Upon vaginal examination the uterus was found to be hard, about the size of a small orange, and seemed irregular in contour over the right cornu. The posterior lip of the cervix was soft and bluish. The anterior lip was fairly hard and covered with small cysts. The vaults were clear.

The temperature was 99°, the pulse 88. The respirations were 22.

Examination of the urine was negative. The blood showed a red cell count of 4,200,000, with a hemoglobin of 80 per cent. The white cell count was 8,750. A Hinton test was negative.

On the third day operation was performed.

Differential Diagnosis

Dr. Judson A. Smith: This case is that of a thirty-six year old housewife. If I abstract the story of her bleeding it is briefly this: She missed a period and then approximately four weeks after that, having attempted to abort herself, she had a period of bleeding which lasted nine days during which she passed no clot or tissue. Approximately four weeks after that she had a two-week period of bleeding which lasted until admission.

The physical examination is negative except for enlargement of the uterus.

The fact that this patient has missed a period immediately suggests, at least to an obstetrician, that she had become pregnant. The history shows that at least during the early part of her married life she had normal fertility and we can deduce from her record that her periods were very regular. Missing a period in a woman of that age, with that history, is strong presumptive evidence of pregnancy. If we suppose that she had become pregnant, what possible causes are there for bleeding? The first thing that occurs to us, and certainly the most probable cause, is that she has an abortion, either incomplete or of the missed abortion type, the so-called blighted ovum. That assumption would fit with practically everything in the history. If she has a blighted ovum of a month or six weeks' duration, at the time of this admission the uterus would be the size described here. Furthermore, it is hard, which is consistent with a blighted ovum because once the ovum is dead the uterus loses the softness that is characteristic of a normal pregnancy. The absence of pain and cramps is no argument either way, and I think the blighted ovum in the uterus would explain the whole situation.

There are some possibilities we might keep in mind even if the patient were pregnant. She might have a mole. The facts against mole are that her uterus is not any bigger than it is—moles grow rapidly as a rule—it is firm, whereas a uterus with a mole is commonly soft. She might have chorionepithelioma, with or without mole. It is a far-fetched diagnosis but one to be kept in mind. I suppose any case of irregular bleeding following a missed period should raise the question of ectopic pregnancy. That ought to be excluded. There is no pain and no tenderness. The vaults are said to be clear, and of course the amount of bleeding is not characteristic of ectopic pregnancy. If she is pregnant at all it seems to me the most probable diagnosis is blighted ovum.

Does she have to be pregnant? Even women who have been conspicuously regular will occasionally miss a period. We might say that she missed it accidentally and then had two very abnormal periods. In that case I think that the first diagnosis to occur to us would be submucous myomata. That would explain the situation very nicely.

If she is not pregnant, enlargement of the uterus and bleeding must be attributed to some form of new growth and certainly myomata are the commonest at this age. Adenocarcinoma of the fundus would be very unlikely at this age, and the amount of bleeding was rather too much for that.

I do not believe there is any way of reasoning yourself to a diagnosis in this case with the evidence that we have. If we knew whether she had any symptoms of pregnancy in the first month or so we would be helped a little bit. We do not know whether she had nausea, vomiting or breast changes. And of course a positive Aschheim-Zondek test would be of help; a negative test would be no good at all.

Dr. Tracy B. Mallory: The Aschheim-Zondek was positive.

Dr. Smith: That puts entirely different light on it. We have to assume that she was pregnant

and that there is still living chorionic tissue in the uterus. I think that increases somewhat the chances that she may have a mole or chorion-epithelioma.

When was that Aschheim-Zondek test done? It must have been done on admission. She was not seen before that.

DR. LANGDON PARSONS: Just before she was operated on.

DR. SMITH: It is possible that just the embryo had died. An Aschheim-Zondek does not necessarily become negative because the embryo is dead. It is a little bit surprising to get a positive Aschheim-Zondek test but I think I would still say that she probably had a blighted ovum and may have a mole or chorionepithelioma. Of course the diagnosis is quite easily settled by curettage and I suppose they did that in this case.

DR. MALLORY: I think we might put it up to you at that stage, Dr. Smith. It was done and was negative.

DR. SMITH: What do you mean when you say that it was negative?

DR. MALLORY: A very small amount of grossly normal endometrium was obtained.

DR. SMITH: Of course there is a very important fact about the case which I do not have, that is exactly how the uterus felt to the man who examined her. It is described as being somewhat irregular.

DR. MALLORY: Perhaps you can help out, Dr. Parsons.

DR. PARSONS: Dr. Smith is perfectly right for I went through the same suffering that he is going through, and it was only on the feeling of the uterus that we decided to operate. We were presented with the Aschheim-Zondek as we faced the patient on the table. The curettage, at least grossly, did not help us much. The uterus was firm and hard, and its outlines appeared normal except for the right cornu where a mass could very easily be felt which seemed to be continuous with the uterus, projecting from it as a round tumor, which extended upward and backward. It was somewhat softer than the rest of the uterus. The only reasons for operating on this patient were that either the Aschheim-Zondek test was wrong, which is impossible, or that there was something wrong with the pregnancy itself.

DR. SMITH: I do not think that it is sensible to assume that the Aschheim-Zondek test is wrong. We had better assume that there was a pregnancy mixed up with it somewhere. The only diagnosis I can see now is an interstitial ectopic pregnancy, which is still alive enough to give a positive Aschheim-Zondek test. What else this mass could be, I do not know.

DR. LELAND S. MCKITTRICK: Would it be conceivable to have a pregnancy in the cornu, not the usual part of the uterus, and bleeding into the uterus instead of into the abdominal cavity, and in that way not have pain?

DR. SMITH: It seems possible, theoretically. I have never heard of a cornual pregnancy that ruptured into the uterus. I would expect that if this happened there would be more bleeding than occurred in this case.

DR. MCKITTRICK: If she was bleeding through the tube into the peritoneal cavity she would have pain.

DR. SMITH: It strikes me as being very unusual to have that type of pregnancy and not have any pain.

A PHYSICIAN: Is there such a thing as cornual pregnancy?

DR. SMITH: Of course there is such a thing. I have never seen one.

PREOPERATIVE DIAGNOSES

Metrorrhagia.
Fibroid uterus.
Endocervicitis.

DR. JUDSON SMITH'S DIAGNOSES

(a) Preliminary, on basis of history:
? Blighted ovum.
? Mole or chorionepithelioma.
(b) Final, after learning of curettage and ether examination:
Interstitial ectopic pregnancy.

PATHOLOGIC DIAGNOSIS

Ectopic interstitial pregnancy.

PATHOLOGIC DISCUSSION

DR. PARSONS: The findings at operation confirmed what was felt on pelvic examination. The mass had a broad base, was connected with the tube just as the tube came off the fundus of the uterus, was soft, bluish, and definitely in the wall of the uterus. We felt that it was interstitial or cornual pregnancy.

When the abdomen was opened we found an ectopic pregnancy in the cornu of the uterus. I might add that there was only a very thin serosal layer between it and the peritoneal cavity. Supravaginal hysterectomy was done. Apparently if this type of ectopic pregnancy does rupture, so many major vessels are torn you never have a chance to do anything about it surgically. Therefore, hysterectomy was done.

DR. MALLORY: We have chosen to put this case up to Dr. Smith serially since it is in such fashion that the surgeon is actually presented with it. One must make an initial diagnosis and determine whether operation is justified. Then only as the results of the ether examination and the curettage become available do the data be-

come sufficient for a final diagnosis which will determine the character of the remainder of the operation.

As Dr. Parsons has said, the uterine wall on the peritoneal surface of this mass was less than a millimeter in thickness so that it probably was a matter of a few days at most, possibly a few hours before rupture into the peritoneal cavity would have occurred. Between the tumor and the cavity of the uterus the wall was thicker, between two or three millimeters in thickness, so that there would seem to be comparatively little danger of rupture there. Exactly why she should have had so much bleeding is not clear.

A PHYSICIAN: Did the mass project into the uterine cavity? Was there any blood in the uterus?

A PHYSICIAN: How do you explain the normal curettings?

DR. MALLORY: The curettings were examined only grossly. I think it is very possible that microscopic examination would have shown decidua-like changes in the cells such as you get often in a tubal pregnancy.

The New England
Journal of Medicine

SUCCESSOR TO

THE BOSTON MEDICAL AND SURGICAL JOURNAL

Established in 1828

Published by THE MASSACHUSETTS MEDICAL SOCIETY under the jurisdiction of the

COMMITTEE ON PUBLICATIONS

R. I. LEE, M.D., *Chairman* HOMER GAGE, M.D.
R. B. OSGOOD, M.D. R. M. SMITH, M.D.
F. H. LAHEY, M.D.

EDITORIAL STAFF

REID HUNT, M.D. STEPHEN RUSHMORE, M.D.
JOHN P. SUTHERLAND, M.D. HANS ZINSSER, M.D.
GEORGE R. MINOT, M.D. BENJAMIN WHITE, PH.D.
FRANK H. LAHEY, M.D. HENRY R. VIETS, M.D.
SHIELDS WARREN, M.D. ROBERT N. NYE, M.D.
GEORGE L. TOBEY, JR., M.D. ROBERT M. GREEN, M.D.
C. GUY LANE, M.D. CHARLES C. LUND, M.D.
WILLIAM A. ROGERS, M.D.

WALTER P. BOWERS, M.D., *Managing Editor*

ASSOCIATE EDITORS

GEORGE G. SMITH, M.D. WILLIAM B. BREED, M.D.
JOSEPH GARLAND, M.D.

THE NEW HAMPSHIRE MEDICAL SOCIETY

PUBLICATION COMMITTEE

CARLETON R. METCALF, M.D. HENRY H. AMSDEN, M.D.
WARREN H. BUTTERFIELD, M.D.

THE VERMONT STATE MEDICAL SOCIETY

PUBLICATION COMMITTEE

WILLIAM G. RICKER, M.D. C. F. DALTON, M.D.
L. H. ROSS, M.D.

SUBSCRIPTION TERMS: *$6.00 per year in advance, postage paid for the United States, Canada $7.04 per year, $8.52 per year for all foreign countries belonging to the Postal Union.*

Material for early publication should be received not later than noon on Saturday. Orders for reprints must be sent to the Journal office, 8 Fenway.

The Journal does not hold itself responsible for statements made by any contributor.

Communications should be addressed to The New England Journal of Medicine, 8 Fenway, Boston, Mass.

THE PREVENTION OF VENTRICULAR FIBRILLATION AND DEATH FROM POISONING BY BENZOL AND CHLOROFORM AND FROM THE EFFECT OF ELECTRIC SHOCK

UNDER the rather misleading title "New Therapy for Heart Disease" there has been recently published in the lay press a summary of some important experiments carried out by Doctors Nahum and Hoff of Yale University. There is among medical practitioners in general so little knowledge of this work carried on by the New Haven investigators that a brief comment on its significance appears advisable at this time and in this place in order that physicians may be cognizant of studies that bid fair to have an important clinical application and at the same time be able to answer the questions of lay patients and friends who have read, with little understanding as a rule, of the marvels of medical progress in the daily newspapers.

The steps in the process of the present study are somewhat as follows. Over twenty years ago Levy[1] working in the laboratory of Thomas Lewis showed that under light chloroform anesthesia ventricular arrhythmias leading to ventricular fibrillation and death could be induced in experimental animals (cats) by conditions which stimulated the heart (such as sympathetic sensory excitation or nervous excitement accompanied by struggling in the earlier stages of chloroform administration) and by equivalent conditions which removed or reduced depressing influences. Full narcosis under chloroform abolished this tendency. It was concluded that in human patients sudden unexpected death under light chloroform anesthesia was due to ventricular fibrillation.

In 1906, 1909 and 1911 Reid Hunt and Taveau[2] had reported their pioneer work in the making and testing on animals of new compounds of choline, among them acetyl choline and trimethyl-(B-acetoxy-prophyl)-ammonium chloride, now generally called acetyl-B-methyl choline chloride. To quote Hunt and Taveau's original words "Some of the compounds with which we have worked are so extraordinarily active physiologically (0.00000001 gram acetyl choline per kilogram animal, for example, causes a fall of blood pressure) and yet are not very toxic that it seems not improbable that some may prove of value in therapeutics." Concerning acetyl-B-methyl choline chloride Hunt and Renshaw[3] have recently written, "We found this new compound to be so stable that it was very active in lowering the blood pressure not only when injected intravenously but when injected subcutaneously and even when given by mouth." They reiterated that the results obtained seemed to offer some promise of clinical usefulness even at that time (1911). Years afterwards this particular compound was "rediscovered" with no reference to the earlier work of Hunt and Taveau. The history of this Hunt and Renshaw recount[3].

Loewi[4] in 1921 showed that the chemical substance acetyl choline, made by Hunt and Taveau in 1909, is actually produced in the body at the vagal (parasympathetic) nerve endings by vagal stimulation, and Cannon and his coworkers[5] in 1931 demonstrated that there is a substance antagonistic to acetyl choline, called sympathin, produced by sympathetic nerve stimulation, and that sympathin and adrenaline have similar properties. For more than a generation physiologists have known that the sympathetic and parasympathetic (vagal) divisions of the autonomic nervous system have opposite effects on various tissues and organs of the body. For example, the heart rate is slowed by vagal stimulation and quickened by sympathetic stimulation.

Also for many years it has been demonstrated by many workers that auricular fibrillation can

be induced by vagal stimulation and by digitalis, a vagal stimulant, in experimental animals and that various stimulants, especially epinephrine (adrenaline) and electric shock, can cause ventricular fibrillation with resulting cardiac standstill and death.

Nahum and Hoff have now advanced the frontiers further in four particulars. In the first place they[6] have shown that benzol acts like chloroform on the heart, partial intoxication with it, in contrast to heavy saturation, tending to induce ventricular fibrillation and sudden death if there is some added stimulus like excitement or effort which adds adrenaline or sympathin to the circulation, and that a fatal issue can be avoided in experimental animals by removal of the adrenal glands and the stellate ganglia.

Secondly, Nahum and Hoff[7] have shown that removal of adrenal glands and stellate ganglia also protects animals from ventricular fibrillation and death when they are exposed to light chloroform anesthesia and electric shocks.

Thirdly, and most significant of all, it has been discovered by these investigators[8, 9], that acetyl choline (or acetyl-B-methyl-choline chloride), the parasympathetic or vagal chemical substance, given to experimental animals protects them from ventricular fibrillation and death even when they are under light chloroform or benzol intoxication and stimulated, as with adrenaline, or when they receive an electric shock of the strength which normally causes fibrillation of the ventricles. The important clinical application which is suggested by these findings is obvious.

On the other hand, and finally, to date, Nahum and Hoff have discovered, as might be expected from work that has gone before, that acetyl choline, like vagal stimulation, can in experimental animals produce auricular fibrillation.

It is of further interest that the effect of acetyl-B-methyl-choline chloride on the heart has already been studied clinically, notably by Ellis and Weiss[10] and by Starr and his associates[11, 12]. The latter investigators found that in forty-seven normal individuals the drug injected subcutaneously produced a prompt and vigorous action with fall in blood pressure, rise of pulse rate (succeeding a brief fall), flushing, sweating, and salivation. The effect of the drug in cases of paroxysmal tachycardia was also studied by Starr. Injected subcutaneously it caused immediate or almost immediate termination of twenty-four paroxysms of tachycardia (seven of auricular or supraventricular type) in nine patients, in whom carotid pressure had generally been an unsuccessful therapeutic measure. Finally, the vascular spasm in Raynaud's disease was controlled temporarily in some cases, and in most cases of hypertension the blood pressure was temporarily reduced.

From the experience of these various workers, therefore, it would seem desirable to try acetyl-B-methyl-choline chloride (mecholin or mecholyl) in some cases with obstinate and severe paroxysmal tachycardia (not subject to asthma), and in the prevention of disaster in cases of benzol or chloroform intoxication. Further studies with the drug are, however, needed to determine the frequency with which there may result untoward reaction from its use.

REFERENCES

1. Levy, A. G.: The exciting causes of ventricular fibrillation in animals under chloroform anaesthesia. Heart. 4: 319, 1912-13.
2. Hunt, R., and Taveau, R. de M.: On the physiological action of certain cholin derivatives and new methods for detecting cholin. Brit. M. J. 2: 1788, 1906.
On the relation between the toxicity and chemical constitution of a number of derivatives of choline and analogous compounds. J. Pharmacol. & Exper. Therap. 1: 303, 1909-10.
The effect of a number of derivatives of choline and analogous compounds on the blood pressure. Hygienic Lab. Bull. 73: 1911.
3. Hunt, R., and Renshaw, R. R.: Further studies of the methyl cholines and analogous compounds. J. Pharmacol. & Exper. Therap. 51: 237, 1934.
4. Loewi, O.: Über humorale Übertragbarkeit der Herznervenwirkung. Arch. f. d. ges. Physiol. 189: 239, 1921.
5. Newton, H. F.; Zwemer, R. L., and Cannon, W. B.: Studies on the conditions of activity in endocrine organs. Part 25. The mystery of emotional acceleration of the denervated heart after exclusion of known humeral accelerators. Am. J. Physiol. 96: 377, 1931.
Cannon, W. B., and Bacq, Z. M.: Part 26. The hormone produced by sympathetic action on smooth muscle. Ibid. 96: 392, 1931.
Rosenblueth, A., and Cannon, W. B.: Part 28. Some effects of sympathin on the nictitating membranes. Ibid. 99: 398, 1932.
6. Nahum, L. H., and Hoff, H. E.: The mechanism of sudden death in experimental acute benzol poisoning. J. Pharmacol. & Exper. Therap. 50: 336, 1934.
7. Hoff, H. E., and Nahum, L. H.: The rôle of adrenaline in the production of ventricular rhythms and their suppression by acetyl-B-methylcholine chloride. J. Pharmacol. & Exper. Therap. 52: 235, 1934.
8. Nahum, L. H., and Hoff, H. E.: The experimental production of ventricular fibrillation by benzol, and its prevention by B methyl acetyl choline chloride. Am. J. Physiol. 109: 78, 1934.
9. Hoff, H. E., and Nahum, L. H.: The nature of ventricular fibrillation following electric shock and its prevention by acetyl-B-methyl choline chloride. Am. J. Physiol. 110: 675, 1935.
10. Ellis, L. B., and Weiss, S.: A study of the cardiovascular responses in man to intravenous and intra-arterial injection of acetylcholine. J. Pharmacol. & Exper. Therap. 44: 235, 1932.
11. Starr, I.; Elsom, K. A.; Reisinger, J. A., and Richards, A.: Acetyl-B-methylcholin (chloride). I. Action on normal persons. Am. J. Med. Sc. 186: 313, 1933.
12. Starr, I.: Acetyl-B-methylocholin (chloride). III. Action on paroxysmal tachycardia and peripheral vascular disease, with discussion of its action in other conditions. Am. J. Med. Sc. 186: 330, 1933.

STREPTOCOCCI AND PUERPERAL INFECTION

It has been surmised for some time that hemolytic streptococci obtained from various sources, such as human infections, bovine infections and milk, were probably different varieties, but attempts to differentiate such groups on the basis of cultural and biochemical characteristics have not led to consistent results. While it is usually true that strains of human origin produce relatively slight acidity when grown in dextrose broth, fail to hydrolyze sodium hippurate and are able to digest human fibrin and that those of bovine origin usually do the re-

verse, certain strains are constantly encountered which cannot be placed in either group.

Serological differentiation employing agglutination as a test method has been unsatisfactory because of spontaneous agglutination so commonly found among streptococci. Several years ago, however, Lancefield[1] described a precipitin test with which she was able to divide all except two of 106 strains of hemolytic streptococci, isolated from various sources, into five distinct serological groups. This reaction was based on the presence in the bacteria of a group specific substance, similar, at least in two of the groups, to the polysaccharide responsible for type specificity among pneumococci. The four main groups were the following: A, those derived from human infections; B, those from bovine mastitis; C, those from a variety of animal sources other than human; and, D, those from cheese.

Confirmation of the accuracy and usefulness of this method of differentiation has been established in a recent paper by Lancefield and Hare[2] in which a group of hemolytic streptococci isolated from the vaginas of women at or about the time of parturition were classified according to the precipitin method. These strains were obtained in England and had previously been classified by Hare[3] and Hare and Colebrook[4] according to biochemical reactions. The latter were able to distinguish the streptococci that were responsible for severe puerperal infection from the majority of the saprophytic type, but in the former group there was an appreciable number of ante partum and post partum strains obtained from patients who either had a normal puerperium or showed signs of only a slight infection. Classification on the basis of precipitin reactions was much more striking. From forty-five cases admitted to the hospital with a diagnosis of puerperal uterine infection, forty-five strains of hemolytic streptococci were obtained, of which forty-four were Group A. From 837 cases cultured routinely post partum in the hospital, eighty-five strains were obtained of which only two were Group A, and one of these cases developed a fatal puerperal infection. Finally, from 855 cases cultured routinely ante partum on the district thirteen strains were obtained none of which fell into Group A.

Interesting and practical corollaries to these findings are the apparent facts that the hemolytic streptococci responsible for severe puerperal infections are not normal ante partum inhabitants of the vagina and that such strains are introduced into the genital tract during or after delivery.

REFERENCES

1. Lancefield, R. C.: Serological differentiation of human and other groups of hemolytic streptococci. J. Exper. Med. 57: 571, 1933.
2. Lancefield, R. C., and Hare, R.: The serological differentiation of pathogenic and non-pathogenic strains of hemolytic streptococci from parturient women. J. Exper. Med. 61: 335, 1935.
3. Hare, R.: Haemolytic streptococci from vagina of febrile and afebrile parturient women. J. Path. & Bact. 38: 129, 1934.
4. Hare, R., and Colebrook, L.: Biochemical reactions of haemolytic streptococci from vagina of febrile and afebrile parturient women. J. Path. & Bact. 39: 429, 1934.

The Massachusetts Medical Society

THE ANNUAL MEETING

The Medical and Surgical Clinics at the Massachusetts General Hospital

When a state society holds its Annual Meeting the committee in charge has the choice of offering clinics at all the hospitals, or of concentrating on one hospital.

Under the former scheme a hospital would often spend a great deal of time developing a program only to find that a distressingly small audience greeted its effort. Therefore several years ago the Massachusetts Medical Society decided to focus its attention on one hospital, and found that this plan was much more successful than the former. As a result this scheme has been continued, the locus being shifted from one hospital to another at each Annual Meeting.

This year there will be a Medical Clinic at the Massachusetts General Hospital on Tuesday morning, June 4, at 10:00 A.M. This Clinic will be conducted while the Section of Surgery is in session at the Hotel Statler. This date and time have been selected so that there will be no conflict between the Hospital Medical Clinic and the Section of Medicine on Tuesday afternoon at the Hotel.

There will be twenty-minute discussions by seven different members of the Staff. Included in these discussions will be the following: Joint Diseases, Cerebral Infarction, Streptococcus Infection, Ablation of the Thyroid, the Treatment of Furunculosis, and the Rôle of Emotion in Precipitating Symptoms. Since this is the only Medical Clinic to be held during the Annual Session it will well repay the effort of those medically minded men who take this opportunity of visiting the Hospital.

At 10:00 A.M. on Wednesday, June 5, in the Surgical Amphitheater, there will be a series of ten-minute talks and demonstrations by various members of the Surgical Staff. This program was arranged at the request of the Committee to accommodate Fellows of the Massachusetts Medical Society who are particularly interested in Surgery and may not care to attend the Medical Sections that forenoon at the Statler. An attempt has been made to cover a wide variety of subjects that will be of especial interest. Several new procedures will be presented with the results obtained to date.

There will be included in this session an evaluation of the passive vascular exercise appara-

tus (Pavaex) devised by Hermann and Reid for the treatment of peripheral vascular occlusion. This method has unfortunately been heralded in the lay press through no fault of the sponsors. Doubtless many members of the profession have been embarrassed by their patients inquiring about "new legs for old".

The new Osteomyelitis Clinic at this hospital will be described and the methods of management of this common and dangerous malady outlined.

Methods of technique on total gastrectomy, ulcerative colitis, common duct stones and pilonidal sinus will be presented.

The treatment of chronic bursitis by a simple method of irrigation and the diagnosis and treatment of the commonly overlooked ruptured intervertebral disc are important.

Other subjects include hyperparathyroidism, essential hypertension, carcinoma of the bladder and demonstrations by the Tumor, Fracture, and Ovarian Dysfunction Clinics.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

SPRAGUE, HOWARD B. A.B., M.D. Harvard University Medical School 1922. Assistant Physician, Massachusetts General Hospital. Visiting Physician, House of the Good Samaritan. Assistant in Medicine, Harvard Medical School, Courses for Graduates. Address: 270 Commonwealth Avenue, Boston, Massachusetts. Associated with him is

ORGAIN, EDWARD S. M.D. University of Virginia Department of Medicine 1930. Research Fellow at the Massachusetts General Hospital 1933-1934. Instructor in Medicine, Duke University, 1935. Address: Duke University, School of Medicine, Durham, North Carolina. Their subject is "Electrocardiographic Study of Cases of Coronary Occlusion Proved at Autopsy at the Massachusetts General Hospital 1914-1934." Page 903.

HUDSON, HENRY W., JR. M.D. Harvard University Medical School 1925. F.A.C.S. Associate Surgeon, Children's Hospital. Assistant in Surgery, Harvard Medical School. His subject is "The Thymus Superstition." Page 910. Address: 66 Commonwealth Avenue, Boston, Massachusetts.

JONES, STEPHEN G. A.B., M.D. Harvard University Medical School 1921. F.A.C.S. Assistant Surgeon to Out-patients; Surgeon to Fracture Clinic; Surgeon to Diagnostic Clinic, Massachusetts General Hospital. Senior Visiting Surgeon, Chelsea Memorial Hospital, Chelsea, Massachusetts. Surgeon-in-chief, Symmes Arlington Hospital, Arlington, Massachusetts. His subject is "Fractures of the Head and Neck of Radius Separation of Upper Radial Epiphysis." Page 914. Address: 270 Commonwealth Avenue, Boston, Massachusetts.

GEORGE, ARIAL W. Sc.D (Hon.), D.M.R.E., M.D. Tufts College Medical School 1906. The Sir James MacKenzie Davidson Decoration 1923. Roentgenologist, Jordan Hospital, Cape Cod Hospital, Attleboro Hospital and Brooks Hospital. His subject is "Relations of Roentgenology to Medicolegal Practice." Page 924. Address: 43 Bay State Road, Boston, Massachusetts.

MATZ, PHILIP B. See This Week's Issue Page 894, issue of May 9, for record of author. His subject is "A Study of Heart Disease Among Veterans. II. Analysis of a Group of Cases as to Age at Onset and Duration of Heart Disability." Page 929.

STILES, PERCY G. S.B., Ph.D. Assistant Professor of Physiology, Harvard University. Assistant Editor, Biological Abstracts. His subject is "Recent Progress in Physiology." Page 933. Address: Harvard Medical School, Boston, Massachusetts.

The Massachusetts Medical Society

SECTION OF OBSTETRICS AND GYNECOLOGY*

THOMAS ALMY, M.D.,
Chairman,
140 Rock Street,
Fall River, Mass.

C. J. KICKHAM, M.D.,
Secretary,
524 Commonwealth Avenue,
Boston, Mass.

SURGICAL INDICATIONS IN ACUTE PELVIC INFLAMMATION

The indications for surgical intervention in acute pelvic disease have been markedly limited since Simpson's paper in 1909 in which he first emphasized the value of conservative expectant treatment over radical surgical procedures. Best available figures show that 75 per cent of tubal disease is caused by the gonococcus which, if left alone, soon dies and has never been found on culture of tubes in which normal temperature of two weeks' duration had been shown in the clinical picture. In nonspecific infections, resolution of the exudate similar to the course in specific infection takes place, but the organism often remains viable for long periods.

There are some few specific pelvic infections in which a spreading peritonitis is noted, progressively coming out of the pelvis and going higher and higher with definite levels of increase of tenderness and peritoneal irritation. These cases are rather few, but certainly there

*A series of short selected articles by members of the Section will be published weekly.
Comments and questions by subscribers are solicited and will be discussed by members of the Section.

is indication to operate and drain without too much exploration to spread the disease further.

Operative intervention to drain a pelvic abscess is sometimes necessary, not as a routine procedure, but to evacuate a pointing abscess or in long drawn out cases in which improvement is not shown.

Removal of a gangrenous appendix during the course of an acute pelvic inflammation is occasionally necessary. Most cases of periappendicitis which develop in the course of a pelvic process quiet down along with the primary cause.

Excessive flowing due to development of retention cysts in the ovary is encountered and may be of such severity as to demand operative interference. This situation is extremely rare, but must be considered.

Intestinal obstruction, secondary to a pelvic process, is seen and must be handled surgically as soon as diagnosis is made.

Every one practicing surgery has been or will be, unfortunate enough to open some patient with acute pelvic inflammation. The question as to the best procedure at that point depends entirely on the character of the patient, her moral tendencies and the likelihood of subsequent infection. If she has been unfortunate and the likelihood of another infection is small, the procedure of choice would be to close without any delay once diagnosis is made.

In general, indications for surgery in acute pelvic inflammation are relatively uncommon. Far more women will recover completely and be able to discharge their normal duties if conservative treatment is practiced.

SECOND ANNUAL POSTGRADUATE MEDICAL EXTENSION COURSE

The following sessions have been arranged by the Committee for the week beginning May 19:

Bristol South (New Bedford Section)

Friday, May 24, at 4:00 P.M., at the St. Luke's Hospital, New Bedford. Subject: Obstetrics and Gynecology (Third Session). Harold E. Perry, M.D., Chairman.

Hampshire

Wednesday, May 22, at 4:15 P.M., in the Nurses' Home of the Cooley Dickinson Hospital, Northampton. Subject: Cardiovascular Disease (Third Session). Robert B. Brigham, M.D., Chairman.

Norfolk (Norwood Section)

Friday, May 24, at 8:30 P.M., at the Norwood Hospital, Norwood. Subject: Cardiovascular Disease (Third Session). Hugo B. C. Riemer, M.D., Chairman.

Worcester (Milford Section)

Thursday, May 23, at 8:00 P.M., at the Milford Hospital, Milford. Subject: Cardiovascular Disease (Second Session). Joseph I. Ashkins, M.D., Sub-Chairman.

A PRIZE FOR CASE REPORTS BY INTERNS IN MASSACHUSETTS HOSPITALS

The Massachusetts Medical Society has provided for the award of a prize of fifty dollars each year for the best written and most comprehensive case report submitted by an intern holding a rotating internship in a Massachusetts Hospital which is approved for intern training by the American Medical Association.

The prize for this year has been awarded to Dr. Joseph C. Edwards of the Springfield Hospital. He has reported a case of Primary Pulmonary Carcinoma in Association with Pulmonary Aspergillosis.

This paper will be published in The *New England Journal of Medicine.*

MISCELLANY

HEALTH IN BOSTON—JULY THROUGH DECEMBER, 1933*

When the Unemployment Census for Massachusetts was taken in the City of Boston as of January 2, 1934, it was decided to include questions concerning health because much has been said regarding the overcrowding of clinics and hospital wards and the decrease in the practice of private physicians. The purpose of this special study was to gather data regarding the illnesses each person had had during the last six months of 1933 to determine, if possible, whether illness was more prevalent among the unemployed and to learn the extent to which free medical facilities were being utilized.

It is recognized that there is seasonal variation in sickness and that the ideal period for study would have been the first four months of 1934. Since, however, the unemployment census was taken as of January, the health information obtained relates to the previous six months. For the purpose of the census, illness was defined "as any sickness which incapacitates a person for two days or more, keeping him or her from work, school, or performance of usual duties; or any sickness that has been diagnosed by a physician, even though the patient is up and about (i.e., heart trouble, arrested tuberculosis, arthritic condition, etc.)". The enumerators asked each family this question: "Has any person been ill in your family between July 1 and January 1?" The statement of the member of the family giving the information about illness was accepted as it was impossible to obtain medical verification. Health information was not secured in all cases, and this fact must be taken into account in the conclusions drawn

*This material was prepared by Margaret H. Tracy, Executive Secretary of the Boston Health League, for the State publication "Census of Unemployment in Massachusetts."

from the material. Sixty-eight thousand and forty-seven (68,047) persons reported seventy-six thousand and seventy-three (76,073) illnesses during this six months' period, approximately nine per cent (8.8) of the persons in the whole population.

The figures were compiled separately for the fourteen Health and Welfare Areas in the City. The number of illnesses reported per person was identical in each Health Area and was 1.1 per cent illness per person.

TABLE 1

Population and Unemployment All Persons by Health and Welfare Areas

	Unemployed		Temporarily Employed		Employed Full Time		Not Seeking Employment			*Number Individuals Reporting Sickness*	
	No.	Per Cent of Total Population	No.	Per Cent of Total Population	No.	Per Cent of Total Population	No.	Per Cent of Total Population	Total Population	Number Reporting Sickness	Per Cent of Total Population
City Totals	88,075	11.5	37,987	4.9	211,049	27.4	432,442	56.2	769,604	68,047	8.8
Back Bay	2,621	7.2	926	2.6	18,246	50.4	14,398	39.8	36,191	1,880	5.2
Brighton	4,553	8.3	1,751	3.2	18,724	34.1	29,937	54.4	54,965	4,690	8.5
Charlestown	3,748	12.8	2,112	7.3	6,304	21.4	17,164	58.5	29,328	2,211	7.5
Dorchester North	12,719	10.4	5,667	4.6	33,049	27.1	70,658	57.9	122,093	10,616	8.7
Dorchester South	7,448	9.6	2,876	3.8	21,564	27.9	45,296	58.7	77,184	6,301	8.2
East Boston	8,666	13.9	3,755	6.0	10,665	17.1	39,279	63.0	62,365	5,272	8.5
Hyde Park	2,997	10.9	1,479	5.4	6,120	22.2	16,906	61.5	27,502	2,458	8.9
Jamaica Plain	3,915	9.0	2,085	4.8	12,421	28.6	25,030	57.6	43,451	3,254	7.5
North End	3,927	16.8	1,583	6.8	4,266	18.2	13,635	58.2	23,411	2,341	10.0
Roxbury	12,834	12.3	5,482	5.2	26,098	25.0	60,016	57.5	104,430	11,077	10.6
South Boston	7,496	13.1	3,071	5.4	12,524	21.8	34,271	59.7	57,362	6,456	11.3
South End	9,786	17.7	3,850	7.0	17,754	32.1	23,903	43.2	55,293	4,825	8.7
West End	3,341	11.8	1,467	5.2	9,730	34.3	13,825	48.7	28,363	2,128	7.5
West Roxbury	4,024	8.4	1,893	4.0	13,535	28.5	28,124	59.1	47,566	4,538	9.5
Island Residents	—	—	—	—	49	50.0	51	50.0	100	—	—

TABLE 2

FINANCIAL CLASSIFICATION OF PERSONS ILL BY WORKERS IN FAMILY BY HEALTH AND WELFARE AREAS

Health and Welfare Areas	*Persons Reporting Sickness*	*Financially Independent*		*One or More Full Time Workers*		*One or More Part Time Workers*		*No Employed Worker*	
		No.	Per cent of Total	No.	Per cent of Total	No.	Per cent of Total	No.	Per cent of Total
City Totals	68,047	4,303	6.3	37,651	55.3	10,195	15.0	15,898	23.4
Back Bay	1,880	289	15.4	1,092	58.1	170	9.0	329	17.5
Brighton	4,690	224	4.8	3,412	72.7	478	10.2	576	12.3
Charlestown	2,211	143	6.5	1,065	48.2	399	18.0	604	27.3
Dorchester North	10,616	603	5.7	6,584	62.0	1,447	13.6	1,982	18.7
Dorchester South	6,301	239	3.8	4,437	70.4	688	10.9	937	14.9
East Boston	5,272	218	4.1	2,176	41.3	1,135	21.5	1,743	33.1
Hyde Park	2,458	138	5.6	1,413	57.5	389	15.8	518	21.1
Jamaica Plain	3,254	171	5.3	2,139	65.7	424	13.0	520	16.0
North End	2,341	95	4.0	765	32.7	539	23.0	942	40.3
Roxbury	11,077	943	8.5	5,268	47.6	1,875	16.9	2,991	27.0
South Boston	6,456	386	6.0	3,305	51.2	1,039	16.1	1,726	26.7
South End	4,825	58	12.1	1,650	34.2	781	16.2	1,810	37.5
West End	2,128	140	6.6	992	46.6	322	15.1	674	31.7
West Roxbury	4,538	130	2.9	3,353	73.9	509	11.2	546	12.0

On the whole a low percentage of sickness was associated with a low percentage of unemployment and conversely a high percentage of sickness was reported in districts where a high percentage of unemployed persons was reported. See tables 1 and 2. For example, in the Health Areas of Roxbury, North End and South Boston a higher percentage of persons reported illness than in the City as a whole and likewise in these same districts a higher percentage of persons reported unemployment or no employed person in the family.

Table 3 shows that during the last half of 1933, 48.1 per cent of the total cases of sickness reported receiving treatment by a private physician. Even in the South End where the percentage of cases going to a physician was lowest, one-third (33.8) had a private physician, while in seven Health Areas (Brighton, Charlestown, Dorchester North, Dorchester South, Hyde Park, Jamaica Plain and West Roxbury) more than fifty per cent of the cases had doctors. With the exception of Charlestown these areas reported less unemployment than the City average.

In view of the fact that clinic attendance has markedly increased during the past four years, it is interesting to see that the highest percentage of cases receiving clinic treatment in a Health Area is 28.1 in the South End, while the North End reports 25.9, Roxbury 22.2, and the West End 23.9. The other Health Areas were below the percentage for the City as a whole (15.6 per cent). Even in the areas reporting the greatest use of clinics, only one case in every four went to a clinic.

Variation in the use of hospital facilities for bed cases was small—one case of illness in every five for the City as a whole reported care in a hospital. If the patient went to the hospital, information is not available as to whether he had his own physician,—it is therefore possible that the percentage of cases having a private physician is even greater than the figures indicate.

The fourth table shows whether the patient paid for his medical care. As is to be expected, cases treated by a private physician show the highest percentage paying for care, but in six Health Areas (Charlestown, East Boston, North End, South Boston, South End and West End) one case in four or five was unable to pay the doctor. In these areas the percentage of unemployment and the percentage of families with no employed worker were higher than in the City as a whole.

As is to be expected, the percentage of paid cases in the out-patient departments of the hospitals is very small, ranging from 4 per cent in South Boston to 77.2 per cent in Jamaica Plain. In Boston 9 per cent of the cases paid for treatment in the clinics.

Slightly more than half the in-patient cases received free medical care. Here again Charlestown, East Boston, the North End, Roxbury, South Boston, South End and West End reported the largest percentages of free care. It should be pointed out that doubtless many persons who paid something did not meet the full cost of their hospital care so that the percentages of pay cases does not represent full pay in many instances.

In general the Health Survey indicates that there is more sickness and more use of free care in areas where unemployment is highest. Despite the fact that the last six months of 1933 were economically as unfavorable as any since the beginning of the present depression, nearly one-half the cases of sickness reported during that period were treated by a private doctor.

TABLE 3

REPORTED CASES OF SICKNESS:—NUMBER RECEIVING NO MEDICAL CARE, CARE BY PRIVATE PHYSICIAN, OR HOSPITAL CARE AS OUT-PATIENT OR IN-PATIENT BY HEALTH AND WELFARE AREAS

Number of Cases of Sickness

Health and Welfare Areas	Cases of Illness	No Medical Care		Private Physician		Out-Patient Cases		In-Patient Cases	
	Total	No.	Per cent	No.	Per cent	No.	Per cent	No.	Per cent
City Totals	76,073	12,201	16.	36,560	48.1	11,883	15.6	15,429	20.3
Back Bay	2,060	361	17.5	980	47.6	228	11.1	491	23.8
Brighton	5,134	621	12.1	3,030	59.0	315	6.1	1,168	22.8
Charlestown	2,402	313	13.0	1,291	53.7	277	11.5	521	21.7
Dorchester North	11,952	1,714	14.3	5,990	50.2	1,751	14.6	2,497	20.9
Dorchester South	6,970	1,004	14.4	3,727	53.5	904	13.0	1,335	19.1
East Boston	5,786	920	15.9	2,868	49.6	832	14.4	1,166	20.1
Hyde Park	2,702	435	16.1	1,483	54.9	244	9.0	540	20.0
Jamaica Plain	3,579	417	11.7	2,104	58.8	325	9.1	733	20.4
North End	2,625	287	10.9	1,180	45.0	658	25.1	500	19.0
Roxbury	12,680	2,365	18.6	4,991	39.4	2,820	22.2	2,504	19.8
South Boston	7,284	1,565	21.5	3,110	42.7	1,100	15.1	1,509	20.7
South End	5,506	908	16.5	1,863	33.8	1,593	28.9	1,142	20.8
West End	2,406	484	20.1	877	36.5	576	23.9	469	19.5
West Roxbury	4,987	807	16.2	3,066	61.4	260	5.2	854	17.1

TABLE 4

CASES OF SICKNESS:—PAY OR FREE TREATED BY PRIVATE PHYSICIAN, IN HOSPITAL OUT-PATIENT DEPARTMENT OR AS A HOSPITAL IN-PATIENT

Health and Welfare Areas	Cases of Sickness Treated by Private M.D. — Total Cases Having Physician	Able to Pay — No.	Able to Pay — Per cent	Unable to Pay — No.	Unable to Pay — Per cent	Cases of Sickness Treated in Hospital Out-Patient Dept. — Total O.P.D. Cases	*Pay* — No.	*Pay* — Per cent	*Free* — No.	*Free* — Per cent	Cases of Sickness Treated as Hospital In-Patients. — Total In-Patient Cases	*Pay* — No.	*Pay* — Per cent	*Free* — No.	*Free* — Per cent
City Totals	36,560	31,751	86.8	4,809	13.2	11,883	1,089	9.2	10,794	90.8	15,429	7,250	47.0	8,179	53.0
Back Bay	980	908	92.6	72	7.4	228	83	36.4	145	63.6	491	258	52.5	233	47.5
Brighton	3,030	2,924	96.5	106	3.5	315	96	30.5	219	69.5	1,168	914	78.3	254	21.7
Charlestown	1,291	988	76.5	303	23.5	277	21	7.6	256	92.4	521	149	28.6	372	71.4
Dorchester North	5,990	5,464	91.2	526	8.8	1,751	115	6.5	1,636	93.5	2,497	1,133	45.4	1,364	54.6
Dorchester South	3,727	3,597	96.5	130	3.5	904	119	13.2	785	86.5	1,335	902	67.5	433	32.5
East Boston	2,868	2,095	73.0	773	27.0	832	70	8.4	762	91.6	1,166	434	37.2	732	62.8
Hyde Park	1,483	1,341	88.6	142	11.4	244	34	13.9	210	86.1	540	330	61.1	210	38.9
Jamaica Plain	2,104	1,997	94.9	107	5.1	325	251	77.2	74	22.8	733	498	67.9	235	32.1
North End	1,180	770	65.3	410	34.7	658	77	11.7	581	88.3	500	189	37.8	311	62.2
Roxbury	4,991	4,267	85.5	724	14.5	2,820	174	6.2	2,646	93.2	2,504	899	35.9	1,605	64.1
South Boston	3,110	2,351	75.6	759	24.4	1,100	44	4.0	1,056	96.0	1,509	437	29.0	1,072	71.0
South End	1,863	1,355	72.7	508	27.3	1,593	71	4.5	1,522	95.5	1,142	247	21.6	895	78.4
West End	877	694	79.1	183	20.9	576	44	7.6	532	92.4	469	193	41.6	276	58.8
West Roxbury	3,066	3,000	97.8	66	2.2	260	67	25.7	193	74.3	854	667	78.1	187	21.9

AN HONOR TO DR. HARVEY CUSHING

In recognition of Distinguished Service Rendered to Humanity, The National Institute of Social Sciences presented the gold medal to four eminent scientists at the annual dinner May 9, 1935. This group consisted of Dr. Harvey Cushing, Cornelius N. Bliss, Hon. Carter Glass, and Dr. George E. Vincent.

The selection of Dr. Harvey Cushing for this honor is especially pleasing to his former associates and friends in Boston, for the great contributions to surgery by Dr. Cushing were, in large part, made while he was professor of surgery at the Harvard Medical School, and surgeon-in-chief at the Peter Bent Brigham Hospital.

DRUG CASES BRING HIGH FINES

Prosecutions for violations of the Federal Food and Drugs Act, terminated during March, brought fines aggregating $2,644, the Food and Drug Administration reports. The highest fine was $1,000, assessed against T. M. Sayman, self-styled herb doctor, who made a fortune commercializing a common plant of the American southwest, called the "soaproot" or "Spanish bayonet".

The T. M. Sayman Products Co., St. Louis, Mo., of which "Doctor" Sayman is practically the sole owner, had shipped in interstate commerce products known as "Sayman's Healing Salve", "Sayman's Vegetable Wonder Soap" and "Sayman's Liniment"; the labels on the liniment claimed it to be a remedy for rheumatism, kidney diseases, pleurisy, tuberculosis, grippe, toothache, croup, cramps, hay fever, earache, deafness, ulcers, snake bite, horse colic and whooping cough. All these claims, the government alleged, were false and fraudulent and therefore in violation of the Food and Drugs Act. Sayman did not contest the government's findings and allegations.

In the early part of his career, Sayman conducted a traveling medicine show.

The Continental Drug Corporation of Alton, Ill., responsible for the shipment of fourteen lots of substandard drug products, such as witch hazel, carbolic acid, paregoric, turpentine, mercurochrome, camphorated oil, cod liver oil and castor oil, was fined $420 on March 12. Products of this nature, commonly used in the home, are standardized by law in the United States Pharmacopoeia and the National Formulary, and variation from the legal standard is punishable as a violation of the Food and Drugs Act when the non-standard articles enter interstate commerce. The purchaser of the common household remedies is in this way protected against cheats and against the danger of false security. There is no comparable Federal standard authority for food, except butter.

The labeling of "Cereal Meal", manufactured by the Cereal Meal Corp., St. Louis, Mo., was admitted to be fraudulent, and the manufacturer was fined $270 for having shipped the "meal" to San Francisco, Denver, Chicago and Boston, at which points it was seized. "Cereal Meal", a mixture of bran and other wheat products, linseed meal and agar agar, was recommended for use in cases of indigestion, chronic appendicitis, and intestinal inflammation and catarrh.

Patent medicine cases terminated during March also included that against the White Herb Mfg. and Remedy Co., Dr. John S. White, proprietor, who had shipped "White's Herb Tonic", a mixture of water, plant extractives, and less than one per cent of alcohol. The product was labeled as a system builder to be used in cases of syphilis, blood poison, rheumatism, kidney and liver troubles, pellagra, indigestion, women's diseases, gallstones, influenza, appendicitis and scrofula, in which conditions it would be worthless. A fine of $50 was assessed.

Russell M. Evans, trading as Etsam Mfg. Co., Hatboro, Pa., was fined $25 for having shipped his product "Etsam" bearing false and fraudulent claims for gallstones, liver, stomach and gallbladder trouble, palpitation of the heart, jaundice, neuralgia, poor circulation, appendicitis and other conditions.

"H. G. C.", labeled as an antiseptic but found to be not antiseptic, brought a fine of $90 to the Acme Chemical Manufacturing Co., Lt., and a fine of $10 to Wm. T. Jay, its promoter, of New Orleans, La. The Scientific Manufacturing Co., Scranton, Pa., shippers of "Pheno-Isolin", were fined $30 when they decided not to contest the government's allegation that "Pheno-Isolin", a mixture of fish oil, rosin, turpentine, camphor, menthol and thymol, would be ineffective in cases of infections, swellings, fever, tetanus, sore throat, sore mouth and gums, coughs, boils, carbuncles, ulcers and pyorrhea, for which it was recommended. "Sanovapor Dexene", bearing claims for diabetes, was also adjudged misbranded, and the Sanovapor Laboratories, Huntington, West Virginia, the manufacturers, were fined $100.

Seizures were instituted during March against the following medicines bearing false and fraudulent therapeutic claims for the conditions indicated: "Allimin" for high blood pressure, arteriosclerosis, kidney and bladder trouble; "Anti-Pyrexol" for ulcers, piles, felons, boils, carbuncles and skin diseases; "Bleachodent Liquid" for pyorrhea; "Booth's Hyomei" for catarrh, bronchitis and hay fever; "Capsicum Salve" for rheumatism and lumbago; "Carbosalve" for sores, wounds and skin infections; "Ditman's Sea Salt" for rheumatism and debility; "Ferro China de Angelis" for anemia, malaria, stomach disorders and general debility; "Ferro China Doria" for anemia, loss of appetite and general debility; "Hall's Canker Medicine" for canker, sore throat, and tonsillitis; "Jaques Little Wonder Capsules" for stomach ailments; "Lucorol" and "Servex Antiseptic Powder" for female disorders; "Mother's Salve" for coughs, croup, catarrh, piles, and skin diseases; "Mrs. Olsen's Valuable Salve" for cuts, boils, old sores, eczema and other skin diseases, varicose ulcers and blood poisoning; "Pyrol" for boils, piles, ulcers and eczema; "Sullivan's Oil", also known as "Sullivan's Indian Oil", for earache, toothache, coughs, catarrh, croup, sore

throat, bronchitis, whooping cough, asthma, influenza, rheumatism and lumbago; "Teenjore Ointment" for eczema, ulcers, piles, psoriasis, boils, ringworm, carbuncles and mange; "Dr. Thacher's Liver and Blood Syrup" for constipation, indigestion, loss of appetite, dyspepsia, blood impurities and palpitation of the heart; "Thymoform" for blood poisoning, sores and wounds; "Unguensalve" for skin diseases; "Artificial Vichy Water Powders" for chronic indigestion, acute stomach trouble, diseases of the liver, kidneys and bowels; "Wilharm's Salve" for boils, carbuncles, felons, abscesses, catarrhs, swellings and all kinds of sores; "Watkins Veterinary Balm" for wounds and skin diseases of livestock; and "Dr. Wright's Wormsol" for worms in chickens and turkeys.—*Excerpts from U. S. Department of Agriculture.*

COMPARISON OF DISEASE INCIDENCE IN CONNECTICUT WITH 1934 AND SEVEN YEAR AVERAGE

MONTH ENDING APRIL 27, 1935

Diseases	1935 Week ending April 6	1935 Week ending April 13	1935 Week ending April 20	1935 Week ending April 27	Average cases reported for week corresponding to April 27 for past seven years	1934 Week ending April 7	1934 Week ending April 14	1934 Week ending April 21	1934 Week ending April 28
Actinomycosis	—	—	—	—	—	1	—	—	—
Amebiasis	—	1	—	—	—	—	—	—	—
Cerebrospinal Men.	1	2	1	—	2	1	2	1	1
Chicken Pox	123	116	123	137	99	71	88	91	127
Conjunctivitis Inf.	7	12	6	—	5	8	—	—	3
Diphtheria	4	5	2	2	14	3	3	1	3
Encephalitis Epid.	—	—	—	—	—	—	—	1	1
German Measles	93	125	149	222	38	1	4	2	—
Influenza	5	7	6	8	6	—	6	2	—
Measles	1191	1779	1065	1263	310	23	55	52	71
Mumps	67	84	91	53	81	118	144	126	131
Pneumonia (Broncho)	34	34	25	33	31	19	30	24	17
Pneumonia (Lobar)	46	58	44	43	37	29	31	45	26
Poliomyelitis	1	—	—	—	—	—	—	—	—
Scarlet Fever	130	105	110	76	82	77	64	91	58
Septic Sore Throat	6	14	9	5	1	1	2	1	—
Smallpox	—	—	—	—	2	—	—	—	—
Tetanus	1	1	—	—	—	—	—	—	1
Trichinosis	1	—	—	—	—	—	—	—	—
Tuberculosis (Pul.)	34	25	41	34	31	28	23	24	16
Tuberculosis (O.F.)	1	1	5	1	3	4	1	2	1
Typhoid Fever	—	1	—	—	—	2	1	1	—
Undulant Fever	2	1	1	—	—	1	—	3	—
Whooping Cough	40	44	46	39	81	95	44	66	81
Gonorrhea	21	16	19	11	31	25	13	21	34
Syphilis	67	47	32	40	55	56	42	48	75

Remarks: No cases of Asiatic cholera, glanders, plague or yellow fever during the past seven years.

MILWAUKEE, WISCONSIN — 1935 CONVENTION CITY FOR THE AMERICAN PUBLIC HEALTH ASSOCIATION

Milwaukee extends a cordial invitation to all public health officials and representatives to come to the annual convention of the American Public Health Association, October 7 to 10, 1935.

The spirit of genuine hospitality is Milwaukee's heritage traced through generations.

The growth of this city from an outpost Indian trading village to its present size as the twelfth most populous city in the United States in less than four generations is one of the marvels of the twentieth century. To-day it stands as the metropolis of Wisconsin and one of the great trading marts of the country.

Milwaukee is known as the most law-abiding large city in the United States. Statistics show less crime

in Milwaukee in proportion to population than any other large American city. This is a great tribute to the character of its citizens, but it is also a tribute to the swift manner in which courts administer justice, and to efficient city administration.

It is easy to get to Milwaukee. Its central location makes it accessible from all parts of the United States at surprisingly low travel rates.

The hotels in Milwaukee are the finest the convention visitor could desire and the accommodations are ample. The rates are reasonable for the service offered.

You will enjoy the convenience of this city. The hotels, theater, shopping and business districts are all within walking distance of each other.

Milwaukee is a typical American city. It is a city of homes and home loving people; pleasant folk whose residential areas are beautiful, in splendor or in moderation all parts of the city bear evidence to its citizens' appreciation of comfort and beauty.

Milwaukee boasts of being the healthiest and safest city in the United States. Repeated awards from the Chamber of Commerce of the United States show statistics not to be equaled by any other city.

Milwaukee is ranked first in the United States in the diversification of its industries.

Plan now to attend the convention, to enjoy the meetings and the attractiveness of a truly ideal convention city. Remember the dates—October 7 to 10, 1935.

CORRESPONDENCE

A COMMITTEE TO INVESTIGATE ABUSES OF MEDICAL CHARITIES

May 4, 1935.

Editor, *New England Journal of Medicine,*

A committee has been appointed by the organized medical profession of the City of Boston as represented by the Suffolk District, the Norfolk District and the Middlesex South District of the Massachusetts Medical Society to investigate instances of abuse of medical charity in the city and endeavor to aid in the correction of any abuse. The executives and trustees of many of the charitable institutions have expressed a willingness to coöperate with this committee and already progress has been made.

It will aid this committee in its work to have specific instances of the abuse of medical charity called to its attention so that it can take up specific instances with the proper authorities, because it is felt that by working on specific problems more results can be obtained than working upon generalities.

The committee sends this letter to the *Journal* with the hope that you will care to publish it so that the medical profession at large will realize that there is such a committee in existence and that this committee will welcome information in regard to the abuse of medical charity in Boston. If any physician knows of such instances, the committee will appreciate it if the details are forwarded to Dr. Channing Frothingham, 1153 Centre Street, Jamaica Plain, Mass.

Sincerely yours,

EDWARD J. O'BRIEN,
CHARLES MALONE,
C. FROTHINGHAM,
COMMITTEE.

LETTERS AND FACTS RELATIVE TO DR. SOUTHARD SOLICITED

Columbia University
College of Physicians and Surgeons
630 West 168th Street, New York
Department of Bacteriology

April 17, 1935.

Editor, *New England Journal of Medicine,*

I have been engaged for the past few months in collecting data, memorabilia, and personal impressions concerning the late Dr. E. E. Southard, at the time of his death (1920) Bullard Professor of Neuropathology, Harvard Medical School, and Director of the Massachusetts State Psychiatric Institute, in order to write his biography. His friends and pupils have nobly responded in so far as I have known how to reach them through personal communication.

I now take this less personal means of reaching others perhaps equally devoted to his memory, whom I have overlooked or not known to have had relations with him, in the hope of reconstructing a still more complete picture of the multiform personality of this gifted man.

Letters from and to him are particularly desired and if forwarded to me will be acknowledged and returned when they have served their purpose. Personal remembrances concerning him have proved to be very vivid and individual and additional ones would be gratefully received.

FREDERICK P. GAY, M.D.

THE EQUILIBRATED SALT DIET

Professional Scientific Service
Publishers
245 Broadway, New York, N. Y.

April 30, 1935.

Editor, *New England Journal of Medicine,*

Our attention has been called to a review of our little monograph entitled "The Equilibrated Salt Diet", which appears in the current issue of your Journal.

Inasmuch as this review contains serious inaccuracies, may we crave space for a brief rejoinder?

None of the distinguished dermatologists who have written on this diet, nor we, ourselves, have alleged that the equilibrated salt (Titro Dietetic Salt) forms "an isotonic solution containing sodium, calcium, potassium, magnesium, chloride, lactate and citrate in proper proportions". The equilibrated salt is a dry mixture whose cation relation is approximately the same as the cation relation of the blood and tis-

sue fluids. The chloride, lactate and citrate content of the mixture does not enter into the theoretical foundations of the diet, which depend upon the cation relation alone.

The real point at issue is not, as conceived by your reviewer, whether "any well-balanced diet will provide the proper supply of cations", but whether continuous ingestion of practically pure sodium chloride (table salt) is capable of deranging the normal cation relation.

That the withdrawal of sodium chloride from a diet unfolds therapeutic effects in various cutaneous disorders is fully established and admits of no dispute. However, recent research has shown that the injurious effects of sodium chloride in such conditions are due to the sodium cation and not to the entire sodium chloride molecule or to the chloride. It has likewise been shown that these injurious effects can be abolished by equilibrating the sodium of sodium chloride with definite proportions of cations which antagonize sodium. The therapeutic effects of this equilibrated salt mixture have been reported on by American and foreign dermatologists and the evidence which we have presented in our monograph should convince the most skeptical that the subject is worthy of serious attention.

The salt problem is inseparably connected with the mineral metabolism. The true significance of the latter is now beginning to dawn and no amount of skepticism will avail against the onward march of this phase of medical science.

We remain, Sir,

Very truly yours,

ROBERT WOLLHEIM,
WALTER H. SCHAUINSLAND, PH.D.

AN APPROVAL OF THE EDITORIAL "TRUTH, SENTIMENT AND MEDICINE"

May 6, 1935.

Editor, *New England Journal of Medicine,*

Just a note to let you know how strongly I approve of the general spirit of the first editorial which appears in the *Journal* May 2.

It seems to me that one of the most unfortunate features in medical practice to-day consists of the separation which has come in between what is called the science of medicine, and the art and practice of medicine. It seems to me that the care of a patient, irrespective of the general approach which may be given, is, or should be, distinctly scientific in its general understanding and whether the help offered is along mental lines, or is produced by expressions of sympathy, or by complex drugs, is immaterial, it all is really a part of the great scientific approach to the healing of the sick.

In the Victorian days, as you speak of them, the physician in those days, — or surgeon, because the surgeons in those days practiced medicine as well as did surgery,—were expected to know all that the so-called science of the day knew, and were to apply it in their daily work with their patients. To-day, unfortunately, the laboratory side of medicine which now has appropriated the term "science," very largely, is expected to dominate the work of the physician, instead of the physician being big enough to take that which is shown in the laboratory findings and use those findings in so far as he is able in caring for his patient.

That which you speak of as sympathy, or understanding, seems to me a real part of what we do for our patients, and if we can relieve an overwrought or tense individual of some unnecessary worry, many times that does as much in quieting the overwrought internal glands as drugs could possibly do, and is entirely in keeping with the science of the practice of medicine.

I am pleased to have you print this note and feel sure that before many years the laboratory will be more definitely for the assistance of the physician in details of his work rather than dominating so much of the work as it does to-day.

Congratulating you, and with best wishes, I am,

Very truly yours,

JOEL E. GOLDTHWAIT, M.D.

372 Marlborough Street, Boston.

ARTICLES ACCEPTED BY THE AMERICAN MEDICAL ASSOCIATION COUNCIL ON PHARMACY AND CHEMISTRY

535 North Dearborn Street, Chicago, Ill.,
April 30, 1935.

Managing Editor,
The *New England Journal of Medicine,*

In addition to the articles enumerated in our letter of April 6 the following have been accepted:

Wm. S. Merrell Company
Diphtheria Toxoid, Alum Precipitated (Refined), ten 1 cc. vials (10 immunizations) package

U. S. Standard Products Co.
Polyanaerobic Antitoxin (Tetanus-Gas-Gangrene), Refined and Concentrated

Yours sincerely,

PAUL NICHOLAS LEECH, *Secretary.*

RECENT DEATHS

BEATTY — FRANKLIN THOMASON BEATTY, M.D., of 139 Beacon Street, Boston, died at his home, April 18, 1935. He was born in Baltimore in 1857, and graduated from the Jefferson Medical College of Philadelphia in 1885. He had practiced in Philadelphia and Boston, but retired several years ago.

He was a member of the University Club, the Algonquin Club, the Country Club of Brookline, the Angelo-American Club of Dresden, Germany, the Bunker Hiil Monument Association, the Bostonian Society, the Sons of the Revolution and the Society of the War of 1812. He was a Mason.

JOHNSON — FRANCIS EMERSON JOHNSON, M.D., of Erving, Massachusetts, died at his home, April 30, 1935, after an illness of several months.

Dr. Johnson was born in Unity, New Hampshire, in 1857, the son of Ezra and Melissa Johnson. His

early life was spent in Claremont, New Hampshire, where he graduated from the high school. He graduated from the Dartmouth Medical School in 1882. He practiced in Erving, Massachusetts, for forty-three years after short periods in Gilsum, New Hampshire, Cooleyville and New Salem.

He joined the Massachusetts Medical Society in 1884 and retired in 1932. In addition to conducting a large practice, he served as town treasurer for twenty-one years and was an officer of the local church. He was an Odd Fellow, Mason and was associated with the Orange banking interests.

The affection and regard of that section were shown by a reception to Dr. Johnson on his seventy-fifth birthday, when prominent citizens of Erving, Orange, Warwick, Wendell, Millers Falls and New Salem assembled in the town hall to do him honor.

Dr. Johnson is survived by his widow, Mrs. Christine (Cuthbert) Johnson; a son, Cuthbert Johnson; a grandson, Emerson Johnson, of Springfield, and a sister, Mrs. Celia Butler, of Philadelphia.

TWOMBLY—Edward Lambert Twombly, M.D., of 222 Beacon Street, Boston, with an office at 144 Commonwealth Avenue, died at his home, May 10, 1935. He was born in Cherry Valley, New York, in 1859, the son of the Rev. Alexander S. Twombly and Mrs. Abigail Quincy (Bancroft) Twombly. He came to Boston early in life, graduated from the Boston Latin School in 1877 and from Yale in 1881. His M.D. degree was conferred by the Harvard Medical School in 1886. He later studied in Berlin, Prague and Vienna, and in the forty-five succeeding years practiced in Boston. He was medical consultant to the Boston Dispensary for more than thirty years and was an instructor at the Tufts College Medical School.

He joined the Massachusetts Medical Society in 1886 and retired in 1932 and was also a Fellow of the American Medical Association. He is survived by his widow, Mrs. Evelyn (Chowne) Twombly, and two brothers, Henry B., of New York City, and Howland T., of Boston.

NOTICES

THE BEAUMONT MEMORIAL HIGHWAY

The citizens of the town of Lebanon and the Beaumont Medical Club have arranged for the dedication of the Beaumont Memorial Highway at Lebanon, Connecticut, on June 1, 1935.

The State of Connecticut has designated the new highway between Lebanon and Willimantic the Beaumont Memorial Highway in honor of Dr. William Beaumont, who was born in Lebanon, November 21, 1785.

The order of exercises is as follows:

12:00 M.: Karl F. Bishop, First Selectman, presiding. Dedication of the Beaumont Memorial Highway, Wilbur L. Cross, Governor of the State of Connecticut; Lieutenant Colonel Robert H. Duenner, Medical Corps, United States Army.

2:30 P.M.: Professor Russell H. Chittenden, presiding. Memorial Address, Dr. Harvey Cushing.

BOSTON UNIVERSITY SCHOOL OF MEDICINE SURGICAL CLINIC AT THE BOSTON CITY HOSPITAL

Friday, May 17, 12-1, Cheever amphitheatre.

Dr. Daniel F. Jones, Consulting Surgeon, Massachusetts General Hospital, New England Hospital for Women and Children, and the Beth Israel Hospital, Boston, will discuss "Diseases of the Rectum with Special Reference to Cancer".

Physicians and medical students are invited.

REMOVAL

Clifford L. Derick, M.D., announces the removal of his office to 520 Commonwealth Avenue, Boston.

REPORTS AND NOTICES OF MEETINGS

CLINICAL MEETING AT THE ROBERT BRIGHAM HOSPITAL

A clinical meeting was held at the Robert Brigham Hospital on Tuesday, April 23. The meeting was opened with the presentation of several charts concerning the diagnosis, classification, and treatment of arthritic conditions. The first case to be presented was a thirty-one year old married woman who, in September 1933, developed a stiff neck and one month later was awakened at night with an acute pain in one of her big toes. The toe was swollen and red, and has been sore ever since. Since that time she has had several joints with a similar condition. In March 1935, an attack of grippe caused the joints to flare up. Physical examination showed the extremities to be cold; and one ankle, one wrist, and several finger joints were swollen. Her sedimentation rate was one hundred and thirteen millimeters, and her blood uric acid was 3.8 milligrams per cent. A urethral smear was negative. X-ray examination of the affected joints showed some periarticular thickening, some irregularity and slight degeneration, and in one joint the space was markedly narrowed.

The findings in this case were felt to be compatible with rheumatoid arthritis, but it was pointed out that the acuteness of the onset in the toes and the glossy swollen appearance of the joints suggested gout. At present she is on a high purine diet to see what effect it will have on her joints. It was pointed out that a similar type of arthritis may follow dieting and a loss of weight. There was much disagreement among those discussing the case as to the prognosis, and it was generally accepted that any attempt to give a prognosis in these cases is futile.

The general treatment of such a case was stressed, and such therapy should be continued for a long time. In general, it can be said that such a patient should be treated as a tuberculosis patient, and the

local joints should be splinted to rest them and later have passive exercise, massage, heat, etc.

The second patient, a twenty-four year old girl, who presented a strong family history of rheumatic fever and who had growing pains at the age of twelve, entered complaining of swelling of her feet. In September 1934 she had a gradual onset, without any previous infections, of a dull ache in her left shoulder on motion. Later her ankle and feet became involved. The symptoms of each joint lasted two or three days and improved on bed rest and salicylates. She was flushed, had a reddened posterior pharyngeal wall, and cryptic tonsils. Her feet were swollen and tender. Except for the fact that the condition in the feet had lasted for several months, the history was typical of rheumatic fever. On one occasion there had been a positive complement fixation test for the gonococcus there in the hospital. Her sedimentation rate was forty-three millimeters, the urethral smear was negative, and a routine complement fixation test was negative. X-ray examination demonstrated a slight periarticular swelling around the finger joints, and the bones of the feet showed some decalcification and soft tissue swelling. The shape of the heart suggested rheumatic fever. It was pointed out that her age is compatible with the type of rheumatic fever that causes rheumatic joints without cardiac complications. Doctor Spink saw this patient at the Boston City Hospital, and feels that her story is very suggestive of rheumatic fever. A high nutritious diet was recommended, as well as plenty of sunshine, and perhaps throat gargles. It was suggested that the feet be put in a cast at right angles.

The third case was that of a twenty-six year old waitress who in July 1933 had an acute onset of pain in the right leg and thigh which was severe enough to send her to bed. Two days later it traveled to the left knee which was swollen for some months. Later the right hand became swollen and painful, so that she found it difficult to get any sleep. The left knee at the time of presentation only moved about twenty degrees without pain and the right wrist was also limited in motion. Physical examination showed the tonsils to be large and boggy and the right ear drum showed a perforation. This case is typical of a gonorrheal arthritis with a sudden severe onset of pain of only a few joints lasting for some time and leaving a residual stiffness. The sedimentation rate was forty-five millimeters per hour. A cervical smear showed many gram negative intracellular diplococci, and the gonococcus complement fixation test was positive, although a second one was negative. By x-ray typical gonorrheal arthritic changes were seen with loss of joint space and some ankylosis without any atrophy of the bones.

Doctor Spink spoke of the experiences of the Boston City Hospital and said that the complement fixation test tends to become negative about three months after the acute arthritis quiets down, and that in the acute arthritis due to the gonococcus this test is positive in ninety per cent of cases. Typhoid vaccine, in amounts of fifty million bacilli, can be given intravenously as a provocative test, and in these cases will often make both the cervical smear and the complement fixation test become positive. He also pointed out that in the large proportion of these cases the knee is involved and that a tap of the knee joint should be done in all cases so that the fluid may be cultured and have a differential count, Wassermann and complement fixation test done on it. Not only is the tap used as a diagnostic procedure at the Thorndike, but also as a means of draining the pus. It may be repeated as often as is necessary. At times an incision is made over the lateral aspect of the joint and the knee is irrigated with about a gallon of hot saline and the incision immediately closed. Of course the primary focus should be treated.

The affected joints should be given exercises within the range of motion that is painless. Splints should support the joints in the lower extremity in the position of weight bearing, if possible, and in the upper extremity with the elbow at about ninety degrees flexion and with the wrist in dorsiflexion. By such treatment complete recovery of articular function is sometimes possible.

WORCESTER DISTRICT MEDICAL SOCIETY

At the Annual Meeting of the Worcester District Medical Society, May 8, the following officers were elected:

President, Dr. William F. Lynch, Worcester.
Vice-President, Dr. Roy J. Ward, Worcester.
Orator, Dr. William A. Bryan, Worcester.
Treasurer, Dr. Edward P. Disbrow, Worcester.
Secretary, Dr. Erwin C. Miller, Worcester.

Councilors on Nominations, Dr. David Harrower, Worcester; Dr. Royal P. Watkins, Worcester—Alternate.

Committee on Funds, Dr. Michael F. Fallon, Chairman, Worcester; Dr. Leslie R. Bragg, Webster; Dr. Edgar A. Fisher, Worcester.

Commissioner of Trials, Dr. Walter P. Bowers, Clinton.

Councilors, Dr. James C. Austin, Spencer, term began 1930; *Dr. Walter P. Bowers, Clinton, term began 1902; Dr. Leslie R. Bragg, Webster, term began 1922; Dr. Frank H. Clapp, North Grafton, term began 1930; Dr. Philip H. Cook, Worcester, term began 1929; Dr. William J. Delahanty, Worcester, term began 1913; Dr. George A. Dix, Worcester, term began 1921; Dr. Ernest B. Emerson, Rutland, term began 1934; Dr. George E. Emery, Worcester, term began 1920; Dr. Michael F. Fallon, Worcester, term began 1916; Dr. Homer Gage, Worcester, term began 1906; Dr. James J. Goodwin, Clinton, term began 1921; Dr. David Harrower, Worcester, term began 1905; Dr. Ernest L. Hunt, Worcester, term began 1918; Dr. Edwin R. Leib, Worcester, term began 1932; Dr. William F. Lynch (President), Worcester, term began 1935; Dr. Arthur W. Marsh, Worcester, term

*Councilor for life by virtue of being a past president of the State Medical Society.

began 1922; Dr. Erwin C. Miller (Secretary), Worcester, term began 1929; Dr. Joseph W. O'Connor, Worcester, term began 1931; Dr. Walter C. Seelye, Worcester, term began 1930; Dr. Edward R. Trowbridge, Worcester, term began 1924; Dr. Frank H. Washburn, Holden, term began 1916; Dr. Royal P. Watkins, Worcester, term began 1927; *Dr. Samuel B. Woodward, Worcester, term began 1902.

Censors, Dr. George A. Dix, Supervising Censor, Worcester; Dr. John J. Dumphy, Worcester; Dr. Lester M. Felton, Worcester; Dr. Thomas F. O'Brien, Worcester; Dr. Harold V. Williams, Whitinsville.

Nominating Committee, Dr. Raymond H. Goodale, Chairman, Worcester; Dr. Gordon Berry, Worcester; Dr. Joel M. Melick, Worcester; Dr. Edward F. Mitchell, Clinton; Dr. Andrew E. O'Connell, Worcester.

Auditing Committee, Dr. Edson W. Glidden, Chairman, Boylston; Dr. Percy A. Brooke, Worcester; Dr. Herbert E. Hedberg, Worcester.

MASSACHUSETTS SOCIETY FOR SOCIAL HYGIENE

Annual Meeting

The Annual Meeting of the Massachusetts Society for Social Hygiene was held at the University Club, Boston, on Tuesday, April 23.

Dr. John H. Stokes, Professor of Dermatology and Syphilology, University of Pennsylvania School of Medicine, was the speaker of the occasion. His subject was "The Doctor, the Public, and the Syphilis Problem."

Dr. E. Granville Crabtree, President of the Society, gave a summary of the work done in the past year. Five hundred and fifty-four lectures on Social Hygiene were given by the four lecturers of the Society reaching 28,000 persons in all parts of the State. Three hundred and thirty-three patients were cared for at the Boston Dispensary Evening Clinics for Syphilis and Gonorrhea, subsidized in part by this Society.

During the year the Society established a Consultation Service after a study of similar services that are being successfully conducted in New York, Philadelphia, and Washington. The Service is supervised by a Sponsoring Committee of fourteen representative physicians, educators, and social workers. It is in immediate charge of a Steering Committee composed of the following: Mrs. Maida H. Solomon, Dr. George Gilbert Smith, Mrs. Eva Whiting White, and, ex-officio, Dr. E. Granville Crabtree.

Institutes for Nurses and Social Workers were held in Springfield and Boston. The Institute in Boston, held on April 22 and 23, was attended by over five hundred social workers.

Dr. Nels A. Nelson, Assistant Director, Division of Communicable Diseases, State Department of Public Health, gave a paper on "How Prevalent Are Syphilis and Gonorrhea?"

Dr. George Gilbert Smith of Boston, President-Elect of the American Urological Association, gave a paper on "What the Social Worker Should Know about Gonorrhea."

Dr. Harry C. Solomon of Boston, Assistant Professor of Psychiatry, Harvard University Medical School, gave a paper on "What the Social Worker Should Know about Neurosyphilis."

Miss Edith Canterbury, Chief of Social Service, Boston Dispensary, and Mrs. Evangeline H. Morris, R.N., Educational Director, Community Health Association, led the discussion of the above addresses.

Dr. Helen I. D. McGillicuddy, Educational Secretary, Massachusetts Society for Social Hygiene, spoke on "Sex Problems among Children and How They May Be Handled."

Lester W. Dearborn, Chief Consultant, Counselling Service, Massachusetts Society for Social Hygiene, spoke on "An Approach to Typical Adult Sex Problems."

The discussion was led by Mrs. T. Grafton Abbott, Educational Secretary, Florence Crittenton League, and Miss Villa T. West, Psychiatric Social Worker, Boston City Hospital.

At the annual election of the Society the following Officers, Directors, and Members of the Executive Committee were chosen:

Officers: Dr. E. Granville Crabtree, President; Mrs. Maida H. Solomon, Vice-President; Miss Elizabeth Ross, Secretary; Mr. William Wadsworth, Treasurer.

Directors: Hon. Sanford Bates, Mrs. Frances Mayer Carter, Mrs. Robert L. DeNormandie, Mr. Charles F. Glueck, Dr. Rudolph Jacoby, Rev. Harry Levi, Mrs. Frank C. Scanlan, Dr. Edward S. Ward, Mr. Robert F. Herrick.

Executive Committee: Dr. Harold L. Leland, Hon. Herbert C. Parsons, Dr. George Gilbert Smith, Dr. Gaylord W. Anderson, Dr. Wilson G. Smillie, Mrs. Eva Whiting White.

THE ESSEX SOUTH DISTRICT MEDICAL SOCIETY

Annual Meeting

The annual meeting of the Essex South District Medical Society was held at the Salem Country Club, Peabody, Mass., on May 8, 1935.

List of officers elected 1935-1936

President: Dr. Hanford Carvel, Gloucester.

Vice President: Dr. J. F. Bradley, Peabody.

Secretary: Dr. R. E. Stone, Beverly.

Treasurer: Dr. Andrew Nichols, III, Danvers.

Commissioner of Trials: Dr. O. C. Blair, Lynn.

Censors: Dr. A. E. Parkhurst (Supervisor), Beverly; Dr. S. N. Gardner, Salem; Dr. S. R. Davis, Lynn; Dr. J. J. Hickey, Peabody; Dr. J. J. Egan, Gloucester.

Nominating Councilor: Dr. C. L. Hoitt, Lynn.

Alternate Nominating: Dr. O. S. Pettingill, Middleton.

Councilors: Dr. C. F. Deering, Danvers; Dr. R. E. Foss, Peabody; Dr. J. F. Jordan, Peabody; Dr. C. L. Curtis, Salem; Dr. N. P. Breed, Lynn; Dr. J. W.

Trask, Lynn; Dr. C. H. Phillips, Beverly; Dr. W. G. Phippen, Salem; Dr. J. F. Donaldson, Salem; Dr. O. S. Pettingill, Middleton; Dr. A. E. Parkhurst, Beverly.

Executive Committee: Dr. J. R. Shaughnessy, Salem; Dr. R. P. Hallett, Gloucester; Dr. C. F. Twomey, Lynn; Dr. Sherman Golden, Beverly; Dr. O. S. Pettingill, Middleton; Dr. C. A. Bonner, Hathorne.

Submitted by the Nominating Committee: Dr. Guy R. Jacobs, Chairman; Dr. J. C. Marchand, Dr. W. W. Babson.

The following, Dr. Hanford Carvel, Dr. R. E. Stone, by reason of their offices, are also Councilors.

The guest speaker was Dr. L. Vernon Briggs of Boston, Mass.

Very interesting medicolegal cases were cited to illustrate and justify the plea that the speaker wished to make, viz., that children, even as young as six to eight years, should be carefully examined physically, mentally and morally, whenever any doubt existed as to their normalcy. In this way, except in true mental deficients, criminal tendencies might be prevented, by appropriate means, other than the usual procedure of expensively punishing the resulting criminal, in later life.

NATHANIEL POPE BREED, M.D., *Reporter.*

FAULKNER HOSPITAL CLINICAL MEETING

The regular clinical meeting was held at the Faulkner Hospital on Thursday afternoon, May 2. One of the cases discussed from the clinical pathological point of view was that of a carcinoma of the rectum which had penetrated into the bladder. A few weeks before death a colostomy was established. For some days preceding death a mass developed in the region of the bladder. The nature of this swelling was not established until the postmortem examination when it was found to be a large abscess which had developed from the ulceration and perforation of the cancer. It was thought during life that it might have been a rapid development of the growth.

The other case was one of unusual interest. Shortly before death the symptoms of intestinal obstruction developed. The patient was known to have a duodenal ulcer with some pyloric obstruction, and two weeks before death, except for the duodenal ulcer and obstructed pylorus there was no evidence of disease along the gastrointestinal tract so far as x-ray studies could determine with the barium given by mouth. A barium enema had not been given. Upon examination of the abdomen just before death there was a large mass in the left lower quadrant which seemed to progress and recede. It was thought that possibly there might have been a markedly dilated stomach and this mass due to the peristaltic waves passing down to the pylorus although the location did not seem to be appropriate for the pylorus. On washing out the stomach, it was found that it was not dilated. Before the patient could be explored she died. At autopsy the large mass felt in the left lower quadrant was found to be an intussusception of the cecum, hepatic flexure and transverse colon into the descending colon extending as far as the sigmoid and pulling with it some of the ileum. There was a carcinoma of the cecum adjoining the attachment of the appendix. Just when this intussusception started is not clear. The patient complained of no pain for the five days preceding death. There was no blood in the bowel movements. There was vomiting each day for the five days preceding death. A duodenal ulcer with some pyloric narrowing was also found at autopsy.

Following the discussion of these cases, Dr. Henry M. Emmons called attention to certain signs and symptoms which occur in the eyes in problems of internal medicine. He spoke of the cyanosis of the retina that occurs in erythromelalgia, and described an instrument for measuring the degree of exophthalmos. He described corneal lesions that may occur as a result of vitamin deficiency. He emphasized the fact that an intracerebral lesion never caused a paralysis of a single eye muscle. Changes in the lens of the eye may be indicative of hypofunction of the parathyroid glands. In dementia praecox he has made a study of the pupils in a series of cases and has found that they are dilated and that this may be diagnostic of the catatonic stage if myopia can be ruled out. If children show paralysis of accommodation he feels that it nearly always indicates a preceding encephalitis. Although toxic amblyopia is usually produced by alcohol or tobacco or a combination of the two he has had some cases when the condition occurred without these etiological factors and he wondered if acetanilid may be another poison which produces the condition as one of his patients was taking a good deal of this drug. He presented a schedule showing the different diagnostic points between metastatic panophthalmitis, orbital cellulitis and the cavernous thrombosis.

Dr. F. William Marlow, Jr., then described a certain type of latent muscle error which his father, Dr. Frank W. Marlow of Syracuse, has been interested in for a number of years. Apparently this latent muscle error can only be detected by putting the eye at complete rest for an appreciable time and then the examination must be made immediately after the eye has been at rest for several days. With this method of examination muscle errors are discovered which are not picked up in the usual routine examination by an ophthalmologist. Patients that have this latent trouble may present three types of symptoms. The symptoms referable to the eye are sensitiveness to bright light and fatigue of the eyes on prolonged use or close work. Another set of symptoms points toward the gastrointestinal tract with indigestion, nausea and vomiting. The third set of symptoms points toward the central nervous system and consists in headache, drawing feeling in the back of the neck and vertigo. Most of these cases are considered psychoneurotic individuals and fail to show any evidence of disease by elaborate

study. There is very apt to be a history of migraine in the family. He described two very striking cases in which the patients had been changed from invalidism due to a variety of these symptoms to entire relief of the symptoms by the correction of this latent muscle error. Apparently the correction does not have to be complete; in fact, better results are obtained if only a certain degree of the error is corrected.

Doctor Marlow emphasized that this condition is not accepted by all as the cause of the symptoms but he and his father have had such striking results that they are convinced the correction of the disturbance in the eye is the curative factor and not due to suggestion.

There will not be any more monthly meetings until the Autumn of 1935.

THE TUFTS MEDICAL SCHOOL ALUMNI ASSOCIATION

The Tufts Medical School Alumni Association has planned a luncheon for Monday, June 3, 1935, at 12:30 P.M. in the President's Room at the University Club. A year ago the Executive Council of the Association decided to arrange a luncheon in connection with the annual meeting of the Massachusetts Medical Society. The speaker will be Dr. Iago Galdston, Executive Secretary of the Medical Information Bureau of the New York Academy of Medicine, whose subject will be "The Economic and Social Aspects of Socialized Medicine—An Analysis of the Socio-Economic Issues Involved."

Any one interested in this problem is invited to attend. Reservations may be made through the Secretary, Dr. Robert T. Phillips, 270 Commonwealth Avenue, Boston.

NEW ENGLAND HEART ASSOCIATION

The regular monthly meeting of the New England Heart Association will be held on May 27 at the Rhode Island Hospital in Providence at 8:15 P.M. Program: 1. Idiopathic Mediocystic Necrosis of Aorta with Rupture. Dr. Robert J. Williams. 2. Migratory Pacemaker. Dr. Frank B. Cutts. 3. Significance of Blood Chemistry Determinations in Cases of Cardiac Edema. Dr. Clifton B. Leech. 4. Comparison of A-V Index with Electrical Axis Deviation. Dr. Henry C. Weyler.

To be read if time permits: Coronary Thrombosis in a Boy of 22. Dr. Frank T. Fulton. Remarks on the Chest Lead in the Electrocardiogram. Dr. Cecil C. Dustin. Two Cases of Cor Pulmonale with Autopsy. Dr. Charles F. Gormly.

The Rhode Island Hospital extends a cordial invitation to members to attend an informal buffet supper at 6:30 P.M. at the hospital before the meeting. Those who expect to come to supper will please notify Dr. Frank T. Fulton at the hospital beforehand.

JAMES M. FAULKNER, M.D., *Secretary.*

HARVARD MEDICAL ALUMNI ASSOCIATION

The Annual Meeting of the Harvard Medical Alumni Association will be held in Parlor D, Hotel Statler, on Monday, June 3, at 12:30 P.M. A luncheon will follow the business meeting. Price $1.00.

VERNON P. WILLIAMS, M.D., *Secretary.*

THE TRUDEAU SOCIETY

ANNUAL MEETING

The annual meeting of the Trudeau Society will be held at the Lakeville State Sanatorium on May 21 at 4 P.M. The speaker will be Dr. Andrew Peters, who will read a paper on "The Place of Heliotherapy in Pulmonary Tuberculosis". A demonstration of the treatment in non-pulmonary tuberculosis, with special emphasis on the technique of heliotherapy, will be presented by the staff of the sanatorium.

MOSES J. STONE, M.D., *Secretary.*

HAMPDEN DISTRICT MEDICAL SOCIETY

A special meeting of the Society will be held in the rooms of The Springfield Academy of Medicine on Monday evening, May 20, 1935, at 8:15 P.M.

Subject: "Medical Economics". Speaker: Dr. M. A. Tighe of Lowell. Dr. James R. Miller of Hartford will also speak.

General Discussion.

H. L. SMITH, *Secretary.*

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, MAY 20, 1935

Tuesday, May 21—

*12 M. South End Medical Club. Office of Boston Tuberculosis Association, 554 Columbus Avenue, Boston.

†2:30-4 P.M. Ward Visit, Massachusetts Eye and Ear Infirmary.

†4-5 P.M. Seminar, Pediatric Laboratory, Massachusetts General Hospital.

Thursday, May 23—

*12 M. Clinico-Pathological Conference. Massachusetts General Hospital.

†12 M. Clinico-Pathological Conference. Children's Hospital.

Friday, May 24—

†12 M. Clinical Meeting of Children's Medical Staff, Massachusetts General Hospital. Ether Dome.

Saturday, May 25—

*10-12. Staff rounds at the Peter Bent Brigham Hospital. Open to practicing physicians.

---

*Open to the medical profession.
†Open to Fellows of the Massachusetts Medical Society.

May 17—Boston University School of Medicine Surgical Clinic at the Boston City Hospital. See page 954.

May 17—The New England Roentgen Ray Society will meet at the Boston Art Club. Dinner 6:45 P.M. Meeting 8:15 P.M.

May 21—The Trudeau Society. See notice above.

May 21—South End Medical Club will meet at the office of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston, at 12 noon.

May 27—New England Heart Association. See notice elsewhere on this page.

June 3—Harvard Medical Alumni Association. See notice above.

June 3—Tufts Medical School Alumni Association. See page 958.

June 10—American-Canadian Medical Golfers Play at Atlantic City. For details write Bill Burns, Executive Secretary, 4421 Woodward Avenue, Detroit.

June 10 and 11—American Proctologic Society will meet at the Marlborough-Blenheim, Atlantic City. For information address Frank G. Runyeon, 1361 Perkiomen Avenue, Reading, Pa.

June 11—American Heart Association. The Eleventh Scientific Session will be held from 9:30 A.M. to 5:30 P.M., at the Hotel Claridge, Atlantic City, N. J. The program will be devoted to various subjects on cardiovascular disease. Gertrude P. Wood, Office Secretary, 50 West 50th Street, New York, N. Y.

June 11—American Neisserian Society will meet at the Hotel Claridge, Atlantic City, New Jersey.

June 12 and 13—Academy of Physical Medicine, Annual Meeting, will be held at the Claridge Hotel, Atlantic City, N. J. For further details address: Arthur H. Ring, M.D., Secretary-Treasurer, Arlington, Mass.

June 17-19—The Medical Library Association will meet in Rochester, New York. For information address Miss Frances N. A. Whitman, Librarian, Harvard University Schools of Medicine and Public Health, Boston, Mass.

June 17 to 21—Convention of the Catholic Hospital Association will be held at Creighton University, Omaha, Nebraska. For information address the Most Reverend Joseph Francis Rummel, D.D., Bishop of Omaha.

June 24-28—American Urological Association and Western Branch Society, American Urological Association, will meet at the Palace Hotel, San Francisco, California. For details write Dr. Charles P. Mathé, 450 Sutter Street, San Francisco, California.

June 27-29 Inc.—British National Association for the Prevention of Tuberculosis will be held at Southport, England. Persons desiring further information should write to Miss F. Stickland, Secretary of the Association at Tavistock House North, Tavistock Square, London, W. C. I., England.

July 1-23—University of Freiburg i. Br. will hold a vacation course of the medical faculty. For information address Akademische Auslandsstelle der Universität Freiburg i. Br., Schwimmbadstrasse 8, Germany.

July 22-27—Seventh International Congress on Industrial Accidents and Diseases, Brussels, Belgium. The American Committee of the Congress is under the chairmanship of Dr. Fred H. Albee, New York, for the Section on Accidents, and that of Dr. Emery R. Hayhurst, Columbus, Ohio, for Industrial Diseases. The American delegation to the Congress will sail from New York on July 8 and visit London, Amsterdam, The Hague and Paris, and, optionally, Budapest. Physicians interested in the Congress or in the medical tour in conjunction with it, may address the Secretary, Dr. Richard Kovacs, 1100 Park Avenue, New York City.

October 7-10—American Public Health Association will meet in Milwaukee, Wisconsin. For information address the American Public Health Association, 50 West 50th Street, New York City.

October 21-November 2—1935 Graduate Fortnight of the New York Academy of Medicine. See page 898, issue of May 9.

DISTRICT MEDICAL SOCIETY

HAMPDEN DISTRICT MEDICAL SOCIETY

May 20—Special meeting. See page 958.

BOOKS RECEIVED FOR REVIEW

Psychology and Health. H. Banister. 256 pp. New York: The Macmillan Company. $2.50.

Sex Practice in Marriage. C. B. S. Evans. 128 pp. New York: Emerson Books, Inc. $1.95.

Useful Drugs. A list of drugs selected to supply the demand for a less extensive materia medica with a brief discussion of their actions, uses and dosage. Prepared under the direction and supervision of the Council on Pharmacy and Chemistry of the American Medical Association. Edited by Robert A. Hatcher and Cary Eggleston. Ninth Edition. 203 pp. Chicago: American Medical Association. $.60.

Diseases of the Rectum and Colon and Their Surgical Treatment. J. P. Lockhart-Mummery. Second Edition. 605 pp. Baltimore: William Wood & Company. $10.00.

Female Sex Perversion. The sexually aberrated woman as she is. Maurice Chideckel. 352 pp. New York: Eugenics Publishing Company. $6.00.

Hoeber's Surgical Monographs. Corrective Rhinoplastic Surgery. Joseph Safian. 218 pp. New York: Paul B. Hoeber, Inc. $9.00.

Studies from the Rockefeller Institute for Medical Research. Reprints. Volume 92. 588 pp. New York: The Rockefeller Institute for Medical Research, 1935.

Modern Motherhood. Claude E. Heaton. A book of information on complete maternity care: Prenatal—Delivery—Aftercare. 271 pp. New York: Farrar & Rinehart, Inc. $2.00.

Ideal Health or the Laws of Life and Health. Alexander Bryce. Third Edition. 340 pp. Baltimore: William Wood & Company. $2.75.

The Harvey Lectures. Delivered under the Auspices of the Harvey Society of New York. 1933-1934. Drs. R. E. Dyer, W. Mansfield Clark, and others. Series XXIX. 262 pp. Baltimore: The Williams & Wilkins Company.

L'Examen du Malade. Guide clinique de l'étudiant et du médecin. Médecine, Chirurgie, Obstétrique, Neurologie et Spécialites. P. Delmas, G. Giraud, et al. 318 pp. Paris: Masson & Cie. 30 fr.

Maimonides (The Rambam) The story of his Life and Genius. J. Münz. Translated from the German, with an introduction by Henry T. Schnittkind. Octocentennial Edition: 1935. 238 pp. Boston: Winchell-Thomas Company. $1.50.

Failure of the Circulation. Tinsley R. Harrison. 396 pp. Baltimore: The Williams & Wilkins Company. $4.50.

Diseases of the Heart. John Cowan and W. T. Ritchie. With a chapter on The Ocular Manifestations of Arterial Disease by Arthur J. Ballantyne. Third Edition. 631 pp. Baltimore: William Wood & Company. $9.00.

Names of Surgical Operations. Compiled and arranged by the Western Surgical Association through its Special Committee. Edited by Carl E. Black. 102 pp. St. Paul: Bruce Publishing Company. $3.00.

Wish and Wisdom. Episodes in the vagaries of belief. Joseph Jastrow. 394 pp. New York and London: D. Appleton-Century Company, Inc. $3.50.

Hertzler's Monographs on Surgical Pathology. Surgical Pathology of the Peritoneum. Arthur E. Hertzler. 304 pp. Philadelphia, Montreal and London: J. B. Lippincott Co.

Maladies Infectieuses. A. Lemierre. 406 pp. Paris: Masson et Cie. 60 fr.

Onchocerciasis. With special reference to the Central American form of the disease. Contributions from the Department of Tropical Medicine and The Institute for Tropical Biology and Medicine. No. VI. Part I by Richard P. Strong. Part II by Jack H. Sandground. Part III by Joseph C. Bequaert. Part IV by Miguel Muñoz Ochoa. 234 pp. Cambridge: Harvard University Press.

The Modern Method of Birth Control. Thurston S. Welton. 168 pp. New York: Walter J. Black, Inc. $3.00.

The Doctor's Bill. Hugh Cabot. 313 pp. New York: Columbia University Press. $3.00.

Electrotherapy and Light Therapy. Richard Kovács. Second Edition. 696 pp. Philadelphia: Lea & Febiger. $7.50.

BOOK REVIEWS

Body Mechanics in the Study and Treatment of Disease. Joel E. Goldthwait, Lloyd T. Brown, Loring T. Swaim, and John G. Kuhns. 281 pp. Philadelphia: J. B. Lippincott Company. $4.00.

Goldthwait and his co-authors present their conception of what is, and what is not, good body mechanics and the effects produced by faulty mechanics of the body. Their presentation as to the definition of normal body posture and mechanics is not necessarily the universally accepted one. Their own personal experience over a number of years has developed in their minds certain conceptions of disease, based upon reactions of the body in its entirety to faulty body mechanics. This must be considered as an expression of opinion regarding the development of all manner of diseases whose etiological factors can be traced back by the authors to faulty body mechanics. In support of their view a number of illustrative cases are presented. These range from backache to diabetes, multiple sclerosis and cancer.

"Would that my adversary would write a book" can hardly be applied by anyone to this piece of work, because among the medical profession there is no man, or group, with whom the reviewer is acquainted who offers upon a purely personal interpretation so much of etiological factors, form of treatment, and clinical entities as those which are described here as related to faulty body mechanics.

However, unless a man has duplicated this work with controls and made a study of a group of patients over a number of years, he is in no position to question specific statements of results obtained in the diseases as described by the authors. "The proof of the pudding is in the eating." This book presents an individual interpretation quite in divergence to many accepted and taught principles, facts of anatomy, physiology and factors producing disease; hence the reader may either believe or disbelieve. It is a matter of obtaining with a certain percentage of patients results satisfactory from both the patient's and the doctor's point of view; but the age-old difficulty arises when one attempts to explain in detail how or why certain results were obtained.

In 1922 a book was written whose title is Body Mechanics and Health by L. C. Thomas and Joel E. Goldthwait. This book might be called a handy compendium to the book, Body Mechanics, now being reviewed. Its contents offer a concise description of body structure, body types with their respective potential functional disturbances, and an outline of exercises. The 1934 publication of Body Mechanics offers many statements which, coming from a group directed by Dr. Goldthwait, cannot be entirely disregarded even if one does not agree with them in toto. Obviously the book represents the opinions of these men and their reasons therefor, and no one can fail to respect opinions based upon such clinical experience as this group has had.

Osteomyelitis. Its Pathogenesis, Symptomatology and Treatment. Abraham O. Wilensky. 454 pp. New York: The Macmillan Company. $9.00.

This book, as the title implies, is a monograph on osteomyelitis, which, after an extensive historical review, a consideration of the basic factors such as the anatomy and physiology of bone and the phenomena of bacterial infection, combines in an orderly manner a general discussion of the disease with statistical data from the Mount Sinai Hospital (New York City) and collected sources. There is an extensive chapter on pathology in which the author emphasizes whenever possible that, "The most important and dominating single element in the entire pathological development is the vascular thrombosis." He states, "From clinical evidence, it usually seems to appear that the amount of bone involved in any focus of osteomyelitis is many times much arger at the very beginning of the process than some time later. The physical basis for this phenomenon lies in the relatively large primary involvement of the bone by the embolus-thrombus formation and the accompanying disturbance of the blood supply in the interior of the bone, and in the secondary contraction of the area of bone tissue thus involved because of the development of collateral circulation." Many will question this idea and at times the author seems to lose sight of the fact that there are other factors which determine the distribution and characteristics of the pathological picture.

There are in all nineteen chapters some of which are relatively short. Such headings as "Symptomatology," "Associated Lesions," which includes a discussion of complications such as joint involvement and of sequelae such as disturbance of growth and various deformities, "Roentgenology," "Treatment," and "Outcome" make up the longer chapters.

In his discussion of roentgenology, the author again emphasizes the picture of the lesion as dependent upon the primary vascular occlusion and subdivides the lesions into "Group A," which includes "periosteal foci" and "cortical foci," and "Group B," "nutrient artery foci" which is subdivided into "primary branch," "subsidiary branch" and "terminal foci". Each type of lesion is illustrated with roentgenograms and the diagrams of the vascular lesion. Granting that bacterial seeding in the vascular tree is the primary mechanism, it would seem that the author should give more attention to changes in the bone secondary to the inflammatory reaction and vascular changes secondary to the inflammatory process occurring in relatively rigid walls. The author should produce more evidence in order to

blame the primary vascular lesion as accounting almost entirely for the distribution of the process. Some of the roentgenograms in this section are not particularly commendable; of these one might mention particularly an "X-ray photograph of tuberculosis of the hip" which is anything but clear or suggestive of the picture usually seen in tuberculosis of the hip.

In the treatment of acute osteomyelitis the author suggests that there is no need to hasten in doing an operation. "Other things being equal, in the absence of a demonstrable bacteremia . . . there should be no surgical attack upon the focus of infection in the bone and no operation of any kind unless suppuration develops and gathers in a well-defined abscess. . . . The reason for the adoption of a conservative attitude is the possible avoidance of an operation of any kind." He then points out illustrative roentgenograms of cases unoperated. These are single roentgenograms in all instances and do not give an idea of the course of these patients. Of these, one is a small localized bone lesion (bone abscess) in the lower end of the radius with little density about the margins; another is an extensive lesion of the humerus which was treated for six months for rheumatism. "At the time this picture was taken the symptoms had practically entirely subsided, there was little swelling, and there was perfect motion." The roentgenogram suggests activity if we may judge from the reproduction.

If there are positive blood cultures, he suggests that there is ordinarily no hurry in rushing headlong into an operation. He advises that one should wait in all cases except those with increasing bacteremia. In the latter group, he swings from his attitude of extreme conservatism to one of "radical removal of bone tissue frequently into healthy areas."

In subsequent therapy, he advises that dressing of the wound is neither necessary nor expedient until the time comes to do the secondary suture of the wound. He suggests that usually after ten to fourteen days secondary suture of the wound should be done.

In the treatment of acute osteomyelitis, the author does not suggest alternate therapies and although we believe that a tone of conservatism in the treatment of osteomyelitis is a good one, by and large, we think that his dictatorial attitude may give rise to misconceptions to those not familiar with the treatment of osteomyelitis.

No mention is made that the indicated therapy may vary as to the age of the patient. We are particularly critical of the author's statement that in joint complications, especially in the hip and wrist, that "as long as the suppuration remains in the intact joint capsule, incision is not necessary". In the chapter on prognosis, it would be interesting to know how many cases in the Mount Sinai series were treated by the method outlined by the author.

The final chapters discuss osteomyelitis of certain regions in which the disease is enough individualized to warrant separate consideration, "Osteomyelitis of the Skull," "Osteomyelitis of the Jaws" and "Osteomyelitis of Vertebrae." Much of the material in these chapters, as well as others, has been previously presented by the author in various medical journals.

It is only to be expected that a book of this kind will get into controversial ground and whatever one feels about certain ideas of the author, the book shows a tremendous amount of work, is adorned with an extensive bibliography at the end of each chapter, and is generously illustrated with roentgenographic and histological reproductions and tables.

It is not a "Bible of Osteomyelitis" and should be read with a critical attitude, but anyone interested in osteomyelitis will find this book a distinct addition to his library.

How to Practice Medicine. Henry W. Kemp. 156 pp. New York: Paul B. Hoeber, Inc. $2.50.

The art of medicine is an intriguing subject to many writers and some day someone is going to write an American essay that will complement Gibson's "little attempt to expand John Lock's Ars Medicae"; a dignified and charming piece of work more especially for Englishmen. The author of the book under consideration has hit 'way wide of the mark, because while he uses an easy conversational "you-and-me" style which makes for effortless reading, he addresses himself to a generation of doctors who are leaving the stage, rather than the younger men who are just beginning their active careers. For good or ill, the top hat and flowing moustache as well as oracular pomposity have given way to the attached collar and colored shirt; to the humanizing of the relations of doctor and patient; to the simplified consultation; to the sincere leveling influence of general education and culture. Few of us would care to adopt many of the ideas and attitudes of the author, well-intentioned though they are. The trained office assistant who can manage a typewriter, keep a checkbook straight and do a venipuncture is a far more valuable person than the trimmest maid. One might question whether favorite prescriptions, however dependable, are not better left for the textbook. Likewise one should look elsewhere for advice on prenatal care, pediatrics and the care of old folks. New members of the county society should be urged to take part in the programs even if they do read their papers; often such papers are far more intelligible than the rambling monologues of some of our seniors. One cannot escape the conclusion that Kemp has taken to himself few young confreres who should be, to "elder statesmen" like himself, a never-failing source of enthusiasm and inspiration. And finally, like many medical books this should have been compressed into a pamphlet, when the introduction, by far the best section, would have made at least half the pamphlet.

Definite Diagnosis in General Practice. W. L. Kitchens. 1000 pp. Philadelphia and London: W. B. Saunders Company. $10.00.

This is a new book; a book designed chiefly to simplify differential diagnosis. One cannot help being intrigued by the challenge of this unique book. There are two distinct divisions to the book. Part I presents 506 symptoms of 407 diseases alphabetically arranged. Part II is devoted to the 407 diseases also alphabetically considered, listing a series of symptoms under each disease. By use of the numbers in front of each symptom or disease, one is supposed to be able to arrive at the most likely diagnosis, the method of use being described in the early pages of the book.

The claim of the publishers that "the results you'll get will be truly amazing" appears, to the reviewer, to be exaggerated. For students its use is to be totally deprecated as their clinical knowledge will not be augmented by it.

There are a number of deficiencies present which tend to weaken considerably the value of the book. It appears fair to state that it makes no provision for the diagnosis of disease in its early stages. I doubt whether one could differentiate the various cardiac arrhythmias by its use. The classification of diseases does not conform with the latest one issued, and used by most hospitals. Although purporting to include atypical conditions one is unable to find any data relating to such conditions as abdominal angina and hence nothing to aid in the differentiation of it and gall bladder disease. Abdominal migraine, brain abscess, swelling (edema) of eyelids in nutritional deficiency, ulcers of the esophagus are either omitted entirely or inadequately treated. For example under pain in the thigh (I, 58) no mention is made of bone neoplasms but Raynaud's disease is included, a most unusual symptom. Under hypometabolism is listed only colloid goitre with no mention of myxedema; on the other hand hypometabolism includes adenomatous and exophthalmic goiter and no mention is made of the leukemias or cardiac failure. Boas-Oppler bacilli are stated as being present in gastric malignancy without, however, intimating the underlying obstruction and hence omitting benign obstruction.

These are only a few of innumerable similar points culled from the book. The claim is made that the work is not complete and that space is deliberately allowed to be filled in by the reader. But is this sufficient justification for the omission of so many points; some important, some minor? Aside from the omissions there are also factual errors as describing the large lymphocytes as macrophages (p. 458, I), the presence of myelocytes in aplastic anemia (p. 462, I) and separating anterior poliomyelitis and infantile paralysis as two entities (p. 473, I).

The reviewer cannot agree with Dr. Musser who states in his foreword that the book will "shorten the time period in which the busy doctor could make a diagnosis" and who at the same time also states that it will foster more intensive study of the patients and stimulate collateral reading.

The attainment of skill in differential diagnosis is a true intellectual pursuit and it is axiomatic that the short-circuited path toward knowledge invariably ends in a culdesac.

There are occasions in the course of practice where the germination of an idea may be sufficiently provocative to lead to a reasoned diagnosis and the book contains many such hints. On the whole the reviewer doubts whether the arrival at a diagnosis by the method of mechanical elimination will prove valid.

Studies from The Rockefeller Institute for Medical Research. Reprints. Volume 91. 598 pp. New York: The Rockefeller Institute for Medical Research, 1934.

This group of reprints, covering the recent work of the departments of the laboratory, of the hospital, and of animal and plant pathology, covers a wide range of subjects. Brief reports are included on factors inhibiting or enhancing tumor growth, on the relation of skin lymphatics to burns, and many other subjects. The results of a study of Rhoads, Castle and others in Puerto Rico on the hookworm anemia are presented in considerable detail in this number and bring out the practical importance of treating the anemia first and the parasitism second.

Allergy and Applied Immunology. Warren T. Vaughan. Second Edition. 420 pp. St. Louis: The C. V. Mosby Company, 1934. $5.00.

The fact that a new edition of this book has become necessary in three years is strong evidence of its usefulness and popularity. In this new edition, nothing has been deleted from the original which is reprinted in its entirety.

The new edition has sixty-one more pages than the old. Several small sections have been added and others amplified chiefly to include discussion of the increasing knowledge of contact dermatitis with the importance of patch tests and the great increase in the number and variety of substances to which certain individuals may be sensitive. Several of the hay fever charts have been changed and to great advantage. Finally, an appendix has been added. It gives directions on various practical topics in some detail, a questionnaire for history-taking, a check list of foods, food diaries, symptom records, and a series of suggested menus with the recipes to go with them. The biliography has been increased from 298 to 318 references. It is unfortunate that, as in the first edition, the references in the text are made only by number and do not include the author's name.

The book still lacks balance and the arrangement could be simplified and improved. The pollen section is too long and too scientific for the layman, while much of the text is too childish for the doctor. Nevertheless, the book as a whole is very readable and it contains a wealth of practical suggestions to help both the professional and the lay reader. It can be recommended to all.